Duplicate

The Medical Clinics of Chicago

MAY, 1917

VOLUME 2—NUMBER 6

Index Number

411883
4.5.43

PHILADELPHIA AND LONDON

W. B. SAUNDERS COMPANY

COPYRIGHT, 1917, BY W. B. SAUNDERS COMPANY. ALL RIGHTS RESERVED

PUBLISHED BI-MONTHLY (SIX NUMBERS A YEAR), BY W. B. SAUNDERS COMPANY, WEST WASHINGTON SQUARE, PHILADELPHIA.

PRINTED IN AMERICA

RC
39
M38
v. 2
no. 6

Important Announcement

The success attained by the Medical Clinics of Chicago convinced us that the profession would welcome a publication embracing the best clinical instruction of the leading medical centers of North America. Beginning with July, therefore, we shall publish

THE MEDICAL CLINICS OF NORTH AMERICA

and with the current number (May) The Chicago Clinics ceases as a separate publication, being merged into The Medical Clinics of North America.

This publication will be issued six times a year (every other month). Each number will be a handsome octavo volume of approximately 300 pages, devoted exclusively to the work of *one* medical center.

The Baltimore number (July) will be devoted to the work of the Johns Hopkins Hospital.

The Philadelphia number (September) will record the work of the University of Pennsylvania and the Jefferson Medical College.

The New York number will include Columbia, Bellevue, Cornell, and the Postgraduate School.

The Boston number will include Harvard, Massachusetts General Hospital, Boston City Hospital, and Peter Bent Brigham Hospital.

The Chicago number will include the institutions familiar to our subscribers through the Chicago Clinics.

In The Medical Clinics of North America subscribers will receive a postgraduate course of unusual scope, giving them the clinical teaching of the great medical centers of the country.

CONTENTS

PAGE

Clinic of Dr. Charles A. Elliott, *Northwestern University Clinic at Wesley Hospital*
JAUNDICE—ITS CLINICAL INTERPRETATION 1091

Clinic of Dr. Frank Smithies, *Augustana Hospital*
CASES OF CHRONIC ENTEROCOLITIS ASSOCIATED WITH THE PRESENCE OF PROTOZOA IN THE STOOLS 1109

Clinic of Dr. M. Milton Portis, *Cook County Hospital*
CARCINOMA OF THE ESOPHAGUS 1143

Clinic of Dr. Charles Spencer Williamson, *Cook County Hospital*
A CASE OF HEMOPNEUMOTHORAX 1159

Clinic of Dr. Arthur F. Beifeld, *Cook County Hospital*
PERNICIOUS ANEMIA 1175

Clinic of Dr. B. C. Corbus, *Rush Medical College, Central Free Dispensary*
GONORRHEAL ARTHRITIS 1189

Clinic of Dr. Sidney Strauss, *Rush Medical College*
CARDIAC ARHYTHMIA 1199

Clinic of Dr. Ralph C. Hamill, *Northwestern University Medical School*
TABES DORSALIS 1217

Clinic of Dr. Charles Louis Mix, *Mercy Hospital*
PRESENTATION OF A CASE OF CARCINOMA OF THE HEPATIC FLEXURE 1236

Clinic of Dr. Frederick Tice, *Cook County Hospital*
LUETIC INFECTION OF THE LUNGS 1253

Clinic of Dr. Walter W. Hamburger, *Cook County Hospital*
HEMATEMESIS 1265

Clinic of Drs. Isaac A. Abt and David C. Straus, *Michael Reese Hospital (Sarah Morris Memorial Hospital for Children)*
SPONTANEOUS PNEUMOTHORAX DUE TO EMPHYSEMA 1291

Clinic of Dr. Frank Wright, *Northwestern University Medical School*
ACUTE NEPHRITIS FOLLOWING TONSILLITIS 1307

Index to Volume 2 1313

CONTENTS OF VOLUME 2

PAGE

Clinic of Dr. Isaac A. Abt, *Michael Reese Hospital (Sarah Morris Memorial Hospital for Children)*

FEEDING THE NORMAL BABY. BREAST FEEDING ... 113
FEEDING THE NORMAL BABY WITH ARTIFICIAL FOODS. FOOD PREPARATIONS ... 249
INFANTILE PARALYSIS (ACUTE POLIOMYELITIS) ... 463
DISEASE RESISTANCE IN RELATION TO NUTRITION OF INFANTS ... 795
DECOMPOSITION ... 805
PYELITIS ... 895

Clinic of Drs. Isaac A. Abt and David C. Straus, *Michael Reese Hospital (Sarah Morris Memorial Hospital for Children)*

SPONTANEOUS PNEUMOTHORAX DUE TO EMPHYSEMA ... 1291

Clinic of Dr. Arthur F. Beifeld, *Cook County Hospital*

DIFFERENTIAL DIAGNOSIS OF A CASE WITH AN ENORMOUS NUMBER OF NUCLEATED RED CELLS IN THE CIRCULATING BLOOD ... 395
PURPURA HÆMORRHAGICA (WERLHOF'S DISEASE) ... 849
CHLOROSIS ... 963
PERNICIOUS ANEMIA ... 1175

Contribution by Truman W. Brophy, M. D., D. D. S.

ORAL INFECTIONS ... 133

Clinic of Dr. James T. Case, *X-Ray Department, St. Luke's Hospital, Chicago*

THE PRINCIPLES OF FLUOROSCOPY OF THE STOMACH ... 199
BARIUM DIAGNOSIS ... 819

Clinic of Dr. B. C. Corbus, *Central Free Dispensary, Rush Medical College*

THE TREATMENT OF SPECIFIC (GONORRHEAL) URETHRITIS, ANTERIOR AND POSTERIOR ... 1057
GONORRHEAL ARTHRITIS ... 1189

Clinic of Dr. Arthur R. Edwards, *Michael Reese Hospital*

THE USE OF DIGITALIS ... 1

Clinic of Dr. Charles A. Elliott, *Northwestern University Clinic at Wesley Hospital*

JAUNDICE—ITS CLINICAL INTERPRETATION ... 1091

Clinic of Dr. Joseph C. Friedman, *Michael Reese Hospital*

CHRONIC DIARRHEAS ... 343
THE DIAGNOSIS AND TREATMENT OF CHRONIC CONSTIPATION ... 1071

Clinic of Dr. Walter W. Hamburger, *Cook County Hospital* PAGE

The Modern Medical Treatment of Chronic Ulcer of the Stomach and Duodenum ... 419
Achylia Gastrica ... 691
Hematemesis ... 1265

Clinic of Dr. Ralph C. Hamill, *Northwestern University Medical School*

Presentation of a Case of General Paresis ... 65
Manic-depressive Insanity or Recurrent Melancholia on a Basis of Dysthyroidism ... 75
Peripheral Neuritis on a Specific Basis ... 81
A Typical Case of Basedow's Disease ... 85
An Unusual Case of Multiple Sclerosis ... 271
External Ophthalmoplegia Due to Disease of the Pons ... 279
Presentation of a Case of Progressive Muscular Atrophy Due to Syphilis of the Anterior Horn Cells ... 289
A Case of Beginning General Paresis ... 298
Acute Anterior Poliomyelitis ... 489
Some Considerations of the Problems of Psychiatry ... 707
Acute Disseminated Myelitis and Acute Syphilitic Meningomyelitis ... 721
Paralysis Agitans ... 949
Tabes Dorsalis ... 1217

Clinic of Dr. Herman L. Kretschmer, *Presbyterian Hospital*

The Treatment of Chronic Colon Pyelitis by Pelvic Lavage ... 567
Fulguration Treatment of Bladder Papillomata ... 941

Clinic of Dr. Charles Louis Mix, *Mercy Hospital*

Staphylococcic Osteomyelitis Developing as a Result of Infection from Teeth ... 89
Presentation of a Case of Paraphasia ... 97
Pleurisy and Gastric Spasm: The Morphin Habit Subsequent to Lead Colic ... 375
A Case of Myelogenous Leukemia ... 384
Two Cases of Primary Pernicious Anemia ... 509
Gastric Ulcer with Intervals of Latency. History of Repeated Gastric Hemorrhages ... 761
Duodenal Ulcer and Complications ... 769
Duodenal Ulcer: Attempt at Perforation into the Gall-bladder Followed by Gastrojejunostomy and Recovery ... 777
Pyloric Stenosis and Cholelithiasis ... 785
Bacillus Aërogenes Capsulatus Infection of the Intestines ... 1007
Aneurysm of the Aorta ... 1017
Presentation of a Case of Carcinoma of the Hepatic Flexure ... 1236

Clinic of Dr. M. Milton Portis, *Cook County Hospital*

Vomiting ... 53
Syphilis of the Stomach ... 359
Carcinoma of the Rectum ... 731
Carcinoma of the Esophagus ... 1143

Contribution by Dr. William Allen Pusey, *St. Luke's Hospital* PAGE

Some Cases of Eczema from External Irritation 533

Contribution by H. H. Schuhmann, M. D., D. D. S., *Chicago*

The Relationship of Oral Foci of Infection to Systemic Diseases. 991

Clinic of Dr. Frank Smithies, *Augustana Hospital*

Cases Illustrating Spasm at the Cardia and Cardiospasm Associated with Diffuse Dilatation of the Esophagus 607

Retroperitoneal Sarcoma 1037

Cases of Chronic Enterocolitis Associated with the Presence of Protozoa in the Stools 1109

Clinic of Dr. Sidney Strauss, *Rush Medical College*

Cardiac Arhythmia 1199

Clinic of Dr. Solomon Strouse, *Michael Reese Hospital*

Diabetes and Surgery; Diabetes and Pregnancy. The Treatment of Diabetic Acidosis 37

Diabetes in the Young 327

A Case of Renal Glycosuria 339

The Diagnosis of Early Active Pulmonary Tuberculosis 745

Inanition in the Treatment of Diabetes Mellitus 999

Clinic of Dr. Frederick Tice, *Cook County Hospital*

Some Cases of Diabetes Mellitus with Complications 13

Diabetes Mellitus with Acidosis 29

Carcinoma of the Head of the Pancreas 303

Chronic Bronchitis, Emphysema, and Marked Cyanosis. Consideration of Congenital Heart Lesions. Other Causes of Cyanosis 311

A Case Presenting Addison's Syndrome 547

Gangrene of the Lung: with Special Reference to Treatment. 557

A Pericardiomediastinitis Secondary to an Acute Plastic Pericarditis 657

Pulmonary Abscess Following Delayed Resolution in a Croupous Pneumonia 661

Rectal Stricture Following Operation for Hemorrhoids 671

An Abdominal Aneurysm 673

A Case of True Amebic Dysentery 679

Anaphylaxis 863

Luetic Infection of the Lungs 1253

Clinic of Dr. Charles Spencer Williamson, *Cook County Hospital*

A Mediastinal Tumor—Probably Hodgkin's Disease 163

Multiple Tubercular Serositis 171

A Case of Bronchiectasis (Unilateral) 175

Recurrent Thrombo-ulcerative Endocarditis 181

A Case of Intermittent Claudication 187

A Case of Cerebrospinal Meningitis 193

Clinic of Dr. Charles Spencer Williamson (*Continued*) PAGE

A Case of Acute Miliary Tuberculosis ... 221
A Case of Syphilis of the Liver ... 237
Polycystic Kidneys ... 577
A Case of Recurrent Endocarditis with Cerebral Embolism ... 593
An Atypical Case of Gout ... 603
Splanchnoptosis ... 631
Three Cases of Pericarditis ... 907
Case I.—Purulent Pericarditis (Pyopericardium), with a Discussion of Pericardiotomy by Dr. Kellogg Speed ... 907
Case II.—Hemorrhagic Pericarditis, with Acute Septic Endocarditis ... 918
Case III.—Serofibrinous Pericarditis ... 927
A Case of Hemopneumothorax ... 1159

Clinic of Dr. Frank Wright, *Northwestern University Medical School*

Acidosis ... 681
Two-hour Renal Test ... 1029
Acute Nephritis Following Tonsillitis ... 1307

Clinic of Dr. Joseph Zeisler, *Northwestern University Medical School*

Etiology and Treatment of Acne ... 317

Index to Volume 2 ... 1313

The
Medical Clinics of Chicago

VOLUME 2 NUMBER 6

CLINIC OF DR. CHARLES A. ELLIOTT

NORTHWESTERN UNIVERSITY CLINIC AT WESLEY HOSPITAL

JAUNDICE—ITS CLINICAL INTERPRETATION

Modern Conception of the Hemoglobin Derivation of the Bile-pigments. Qualitative Tests for Bile-pigments and Acids. Fragility Test of Chauffard and Widal.

Physiology of the Liver. Theories as to the Formation of Urobilin. Physiologic Cholemia.

Classification of Jaundice. Illustrative Cases:

(1) Hepatogenous Jaundice Due to Common Duct Obstruction.

(2) Jaundice Associated with Cirrhosis of Liver.

(3) Catarrhal Jaundice of Infectious Type.

(4) Chronic Hemolytic Icterus—Family Type. Acholuric and Choluric Crises.

(5) Dissociation Jaundice in Pernicious Anemia.

THE conception of jaundice has undergone remarkable changes during the past few years. Many new ideas, based upon reliable experimental data, have recently been added to our knowledge of the chemistry and physiology of the bile-pigments and acids. These facts are of exceptional value to the clinician in the interpretation of the phenomena associated with jaundice as observed at the bedside. The present clinic will be devoted to a consideration of the clinical application of some of these phenomena.

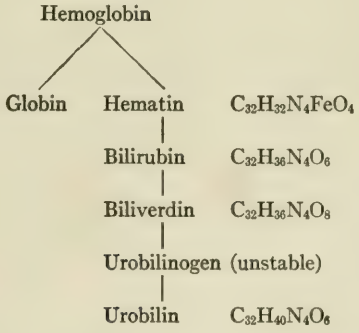
Jaundice is widely observed in clinical medicine. If one includes under the term "jaundice" the modern conception of the physiology of the bile-pigments, their origin, function, and

elimination, together with the possible abnormal manifestations under pathologic conditions, it is a subject of clinical importance ranking with such major subjects as nutrition, respiration, and immunity. Jaundice is a poor term for such wide application, particularly since its use has commonly been restricted to the idea of obstruction to the flow of bile through the biliary passages. With the better understanding of some of the other conditions that result in jaundice the term has come to include much more than the idea of reabsorption of bile from obstructed passages. However inadequate the term may seem, it is the only one at our disposal. Until another word is available, the term jaundice must be understood to connote the conception that bile-pigments are primarily blood-derived pigments excreted by the liver together with the bile-salts and other products of liver activity, the former (pigments) being relatively non-toxic, the latter toxic. The toxic phenomena produced by the reabsorbed bile-salts and excretory products are incidental to, but not fundamentally associated with, the bile-pigment elimination, and occur only in obstructive jaundice. Under this head must be included the various manifestations commonly known as cholemia. There is hardly a disease in which jaundice may not play a rôle either as a primary manifestation or as a result of some complication. Indeed, in a broad sense the phenomena associated with the building up and tearing down of the hemoglobin molecule—a normal process—may be said to be closely related to jaundice. Any disturbance or exaggeration of this normal mechanism is apt to produce jaundice.

The history of the development of the modern conception of jaundice reads like a novel. The older German school, represented by Virchow, Stadelmann, Vierordt, and Minkowski, held that there could be no jaundice without the agency of the liver. The later French school, represented by Widal and Chauffard, developed the idea of hematogenous jaundice. Lyon-Caem (1910), in France, and Whipple and Hooper (1916), in America, finally established beyond any possibility of doubt that bile-pigment is derived, wholly or in part, from the break-

ing up of the hemoglobin molecule. Eppinger (1913), in Vienna, has emphasized the great importance of hematogenous icterus, and the part played by the spleen in the elaboration of the bile-pigments. Syllaba (1904) first recognized the icteric factor in pernicious anemia, and finally the idea of dissociation jaundice has been emphasized by Lemierre (1914) in France, and by Hoover and Blankenhorn (1916) in America.

There has been much work done on the chemistry of the bile-pigments, notably by Hans Fischer (bilirubin), Frederich von Mueller (urobilin), and Neubauer (urobilinogen) in Germany, and by Wilbur and Addis (urobilin) in America. The knowledge of the physiology of the bile-pigments is now being rapidly developed, notably by Whipple and Hooper in America, but as yet is far from complete. Their hemoglobin derivation may be roughly represented by the following diagram:



Hematin contains the iron molecule, bilirubin, no iron. Bilirubin is crystallizable and hence may be obtained in the pure state. Urobilinogen and urobilin are names applied to groups of bodies, the former very unstable, which are closely related, but have not been obtained in the pure state. Hematin, hemosiderin, hematoidin, hematoporphyrin, bilirubin, biliverdin, bilifuscin, urobilinogen, urobilin, and stercobilin are all closely related, some are identical, and all are chemically and physiologically related to chlorophyl.

Of the tests for bile-pigments and acids that are available it is striking that the qualitative tests are quite satisfactory,

while the quantitative tests are entirely unreliable. Bile-pigments give beautiful and characteristic color reactions, and some of them (urobilin and urobilinogen) show characteristic spectral absorption bands. The quantitative tests so far devised are crude and unreliable, depending either upon the conversion of bilirubin into biliverdin with acid alcohol, and then the estimation colorimetrically against a known standard, or, as in the case of urobilinogen and urobilin, the determination of the point of extinction of the characteristic spectroscopic absorption bands by successive dilution of the specimen.

Both these quantitative methods are open to the criticism that the bile-pigments, as extremely labile substances, are constantly changing and are exquisitely sensitive to oxidization. Sunlight and acid, even in weak dilutions, readily alter or destroy bile-pigment in solution. The quantitative estimation of the bile-pigment in the duodenal content would be very desirable as a direct estimate of the amount of blood destruction in a given case, but the same criticism of the proposed methods applies. The qualitative tests for bile-pigment are satisfactorily applied to the duodenal content obtained through a duodenal tube.

Of the many tests and innumerable modifications that are available for clinical purposes the following may be recommended:

For *bilirubin* in the blood-serum, urine, or duodenal content the Gmelin test is satisfactory. When blood-serum is layered over nitric-nitrous acid a play of colors, light blue predominating, appears almost immediately in the rather wide coagulum. The test is fully as satisfactory as when applied to urine. It is said to be sensitive in 1 : 80,000 dilution of bile-pigment.

For *urobilin* the fluorescence of the zinc salts as performed in Schlesinger's test may be considered satisfactory and is thought to be sensitive in 1 : 2000 dilution. Bilirubin when present in large amounts should be previously removed with calcium chlorid and the filtrate extracted with amyl alcohol, using the extract to make the test.

For *urobilinogen* the Ehrlich aldehyd reaction may be used.

The spectroscope should be used to confirm the presence of urobilinogen and urobilin which give characteristic absorption bands. Bilirubin gives no characteristic band.

For the *bile acids* and *salts* the Pettenkofer test is quite satisfactory. Although not specifically a test for these bodies, under the conditions present in jaundice there are not likely to be found any interfering substances which would produce the furfural reaction.

The *fragility* test of Chauffard and Widal for decreased resistance of the red blood-cells to hypotonic salt solution should be mentioned in this connection. It is best performed under sterile conditions, as any bacterial contamination is likely to interfere materially in the reading. Successive dilutions of sterile salt solution (0.6 to 0.24 per cent.) and defibrinated unwashed red blood-corpuscles are used. The test may be read in two to twenty-four hours. It is thought that red cell fragility is present only in hemolytic jaundice.

Physiology of the Liver.—It is safe to say that the bile-pigments are all primarily blood-derived pigments representing different stages in the cleavage of the hemoglobin molecule. Whether this change takes place in the tissues, the circulation, the spleen, or the liver is not certainly known. It probably takes place under varying conditions in all these locations. Under normal circumstances the liver acts as an excretory organ in the elimination of bilirubin, but whether the pigment is carried to the liver preformed, or is elaborated by the liver cells, is not entirely clear. Certain clinical observations indicate that urobilin at least may be formed directly from hemoglobin in the blood-stream, as occurs in the crises of chronic hemolytic icterus, when a large part of the available blood may be destroyed within a few hours, with the liberation of great quantities of urobilin and apparently no bilirubin. Such massive cleavage of the hemoglobin molecule probably is extrahepatic in origin.

Because of the peculiar structure of the liver lobule it is probably easy for the bile to be forced back between the liver cells from the biliary to the vascular capillaries and to enter

the general circulation when obstruction of the bile tracts occur. It is to be observed in this connection that the pressure in the biliary radical must be extremely low since the flow of bile is readily stopped.

Bile-pigment is normally excreted as bilirubin by the liver. After entering the intestines it is readily reduced to urobilinogen and then to urobilin. Urobilin, or its chromogen, is partly absorbed by the intestinal mucosa and enters the blood-stream, from which it escapes wholly or in part through the kidney filter. Since bile-pigment is eliminated from the body it is probable that the eliminative change described is an excretory process.

The bile acids (glycocholic and taurocholic) are excreted by the liver and serve in the process of digestion. The "circulation of the bile"—*i. e.*, the cycle of excretion by the liver, reabsorption by the intestine, and return to the liver through the portal circulation—has been abundantly proved and is evidently a conservative process. No comparable circulation of the bile-pigments has ever been demonstrated (Whipple and Hooper). It is reasonable to assume from this that these two phenomena, excretion and secretion, represent two of the manifold functions of the liver.

The theories as to the formation of urobilin are numerous. Among them may be mentioned the intestinal, renal, and hemolytic. The intestinal theory is based on the observation that while urobilin is constantly observed in the normal urine, it disappears with complete obstruction of the common bile-duct. The renal theory is based upon the fact that urobilin is constantly found in large quantities in the urine in certain diseases associated with great destruction of red blood-cells, as chronic hemolytic icterus and pernicious anemia, but cannot be demonstrated in the blood-serum of the patients. The hemolytic theory is based on the detection in chronic hemolytic icterus of great quantities of urobilin in the urine during the periods of hemolytic crises.

The bile-pigments are believed to be relatively non-toxic, while the bile-salts, on the other hand, are believed to be very toxic, producing depression of the central nervous system,

paralysis, coma, tetanizing agents, icteric necrosis of the liver, and albuminuria. It must not be forgotten that there are probably other products of liver secretion which are reabsorbed along with the bile-pigment and acids when the common bile-duct is obstructed. Probably "cholemia," in the restricted sense, is as much the result of other concomitant conditions, such as acidosis and absorption of other toxic substances, as to the reabsorption of pigment or bile acids.

A classification of jaundice attempted at the present time is certain to be inaccurate and incomplete because many of the fundamental facts concerning the chemistry and physiology of the bile-pigments are unknown. The following classification, however, may be made as a working basis for study:

Classification of Jaundice

From a pathologic standpoint (after Gauvenet):

I. *Hepatogenous.* (Resorption of liver excretory products; bilirubinemia, cholemia).
   1. Obstruction of ducts, *i. e.*, stones, tumors, cysts.
   2. Obstruction of fine bile capillaries (biliary thrombi). Epidemic, infectious, catarrhal.
   3. Damaged liver cells, *i. e.*, phosphorous, chloroform, acute yellow atrophy.
   4. Passive congestion.

II. *Hematogenous.* (Retention in blood-stream of débris of hemolysis; urobilinuria, acholemia.)
   1. Blood alterations:
      (*a*) Icterus neonatorum; paroxysmal hemoglobinuria.
      (*b*) Chronic hemolytic icterus (congenital, acquired).
      (*c*) Icterogenous pernicious anemia.
   2. Splenic alterations:
      (*a*) Icteric myelogenous leukemia.
      (*b*) Banti's disease.
      (*c*) Hemolytic and icterogenous splenomegalies (hypertrophic cirrhosis of Hanot).

3. Infections, *i. e.*, syphilis, pneumonia, yellow fever, scarlet fever.
4. Parasites, *i. e.*, paludism, ankylostomiasis.
5. Intoxications, *i. e.*, toluylendiamin, arsenic, saturnia.

From a physiologic standpoint (after Hoover and Blankenhorn):

1. Complete jaundice.
2. Hemolytic jaundice.
3. Hepatic dissociation jaundice.
4. Renal dissociation jaundice.

In considering any classification of jaundice it may be well to emphasize the statement of Gauvenet that *physiologic* cholemia is a real fact and its exaggeration is at the bottom of all types of icterus. Bile-pigments are probably constantly present in the blood-stream and are rapidly and constantly being excreted by the liver and kidneys. It has been known for a long time that urobilin as such, or as its chromogen, is absorbed from the intestinal content and is carried through the blood-stream to the kidneys, where it is excreted. This is a normal process. Whether bilirubin is likewise formed outside of the liver, possibly throughout the tissues wherever hemoglobin is being broken down, and is then excreted by the liver, or whether the liver itself transforms the bile-pigments from the hemoglobin molecule and then excretes the cleavage products in the form of bilirubin, is at present not definitely known, and opinion is divided upon the subject.

Hepatogenous jaundice (cholemic, choluric) presupposes interference with liver function and the retention not only of bile-pigments but of other secretory and excretory products (bile-salts, toxic bodies). These substances are then vicariously excreted by the kidneys, the body being surcharged with relatively non-toxic bile-pigment and other much more toxic products of faulty liver elimination (cholemia).

Illustrative Cases.—Case I.—This patient, a physician fifty years of age, complains of jaundice of five weeks' duration and of loss in weight, indigestion, and itching.

Until four years ago he was in good health. At that time

an infected great toe caused chills, fever, and lymphangitis and kept him in bed for four weeks. For a year afterward he felt below par.

About three years ago pain in the region of the gall-bladder appeared and remained localized three or four days. A transient jaundice developed. One year later a similar though more severe attack came on. The urine was dark, the scleræ were icteric, and colicky pain, relieved by morphin, was localized in the region of the gall-bladder. Except for mild attacks of indigestion he was well until December, 1916.

On December 31st, following a meal of fried oysters, colicky bowel pain, chiefly epigastric, appeared. Bowels did not move. Pain in the right hypochondrium developed and gradually disappeared. A few days later the urine became dark and the skin began to itch. Stools became clay colored, studded with bubbles—sprue-like.

In the second week fever developed, and then chills and drenching sweats, which lasted for two weeks. Then for several days the bowel movements seemed to be in the nature of diarrheal explosions. In the past two weeks the patient has felt better. The jaundice seems less intense, although the stools are still clay colored. They have never contained microscopic blood. The patient has lost 30 pounds in weight.

The mother of the patient died of cancer of the uterus at the age of fifty-four. Syphilis is denied. Repeated Wassermann tests over a period of years, in which the patient's blood was used as a negative control, were negative.

Physical Examination.—This patient is intensely icteric. The skin throughout is of a dark brown-yellow color. The scleræ are dark yellow. He is well nourished. Temperature and pulse-rate are normal. Adenopathy is not present except at the groin. The lungs appear normal except for numerous râles in the apices. The heart outlines and tones are normal. No capillary pulse is present. The area of liver dulness is of normal size. The liver edge extends about 2 cm. below the costal border, is hard—not tender. The gall-bladder is not palpable; the region is firm, but there is no muscle defense.

The splenic area is larger than normal, but the edge is not palpable. Reflexes are normal.

Laboratory Findings.—The laboratory reports are of particular interest. Stools have all been clay colored, with no evidence of bile-pigment, macroscopic blood, or mucus. The benzidin test gives an intensely positive color reaction.

The urine, darkly pigmented, contains large quantities of bile acids and pigments, but no evidence of urobilinogen. A trace of albumen and many granular and hyaline casts are present, but no sugar.

Blood-serum contains no hemoglobin, urobilin, or urobilinogen, but large amounts of bile acids and pigment.

Fragility of red blood-cells, .48 to .26; R. B. C., 4,124,000; W. B. C., 8200.

Fluoroscopic examination showed normal heart, aortic arch, and lungs. The stomach outline, cap, and emptying time were normal. No obstruction or adhesion of stomach, small bowel, or colon was found.

Fractional stomach examination (Rehfuss) indicated no free HCl in the fasting state, but a total acidity of 10. The first trace of free acid appeared one hour after an Ewald meal; a maximum of 30 was reached after one and one-half hours; after two and one-half hours, when the stomach was emptied, the free HCl was 20. The total acidity curve with a maximum of 60 was parallel with that of the free acid.

The duodenal content consisted of clear mucoid material with a faint trace of free acid. No bile salts or pigments were present.

Comment.—This is a case of complete jaundice. Bile-pigments and bile salts are present in great quantity in both the blood-serum and urine, but are absent in the duodenal content. No urobilinogen or urobilin can be demonstrated in the urine. It must, therefore, be a case of complete obstruction of the common duct. As to whether the obstructing agent is a common duct stone or a carcinoma of the head of the pancreas, the two most likely causes of a persistent complete obstruction of the common duct, it is impossible to say. The fact that the

stools were frothy and the patient had a period during which the stools were very loose suggests pancreas involvement either by malignancy or by stone at the ampulla of Vater.

(Since the presentation of this case an operation was performed and a common duct stone (cholesterin) was found with a similar stone in a "strawberry" gall-bladder. Many adhesions were present around the gall-bladder and common duct.)

Case II.—The second patient, a clergyman forty-two years of age, complains of itching and slight jaundice.

Until about one and a half years ago he complained of almost continuous mild stomach distress present for twenty years. Three to five hours after eating the entire body ached, the stomach felt full, there were eructations of gas. These sensations were aggravated by eating and relieved by soda or induced vomiting. More severe attacks, usually associated with headache, appeared at one- to three-month intervals without apparent cause. These attacks lasted twenty-four hours, and left a peculiar sore sensation in the liver region. Headaches were rather frequent. The more severe attacks were associated with gastric and liver symptoms.

In the past one and a half years the stomach symptoms are less marked and only once has headache developed. There is almost constant discomfort in the liver region. The skin is itchy, dry, and scaling. The intensity of itching varies directly with intensity of liver distress. Slight jaundice, chiefly of the scleræ, has been noted at times.

At thirty-two years of age the patient had malaria for three to four weeks. At twenty years he had an obscure general infection. "Grippe" in 1909. A father and a sister died of pulmonary tuberculosis.

The patient is well nourished, anemic, subicteric, but not definitely jaundiced. Heart and lungs are normal. The liver area is larger than normal. The edge is sharp and hard, two fingerbreadths below the costal margin. The spleen cannot be palpated.

Blood-serum contains bile salts.

Red cell fragility, .48 to .36; R. B. C., 4,712,000; W. B. C., 9600.

Urine is normal.

The gastric secretion curve of a fractional test was normal.

Fluoroscopic examination of chest and of gastro-intestinal tract showed normal conditions.

Comment.—This patient represents one of the very common mild types of jaundice. He has frequent attacks of "biliousness" with headache and slight jaundice. While the exact cause and nature of the liver disturbance is not clear, the probability of a cirrhotic change is suggested, supported by the frequent observation of cirrhosis at the necropsy of patients who give a negative alcoholic history.

Diagnosis.—Cirrhosis of the liver.

Catarrhal Jaundice.—The old explanation of catarrhal jaundice as due to obstruction produced by a plug of mucus in the common duct has been discarded. It is a ready hypothesis for the explanation of this form of jaundice, but it fails to explain undoubted cases, which at necropsy have showed no obstruction whatever, but which showed the bile-passages filled with a thin mucoid material, the walls of the ducts glistening and entirely free from inflammatory reaction.

Recently German and Japanese workers have isolated spirochetes from the blood and organs of cases of infectious jaundice, Weil's disease, and of injected guinea-pigs, rats, and mice (Uhlenhuth and Fromme, Huebner and Reiter, Inada, Ido, Hoki, and co-workers). It is suggested that many of the mild cases of jaundice commonly diagnosed "catarrhal" are really of the infectious type.

Illustrative Case.—This patient, twenty-eight years of age, has recently returned from military service on the Mexican border.

He complains of jaundice, light-colored stools, and dark urine. Six days ago the urine appeared darker than usual. Three days later he first noticed that the scleræ appeared yellow. The skin gradually developed a deep brown color. Before this time the patient was apparently in perfect health. There have

been as yet no symptoms of itching or nausea. Yesterday he had diarrhea—six or seven bowel movements. No pain or tenderness anywhere, no previous attack; no jaundice in the family.

Five years ago the patient apparently had a chancre, but no positive diagnosis was made and no systemic treatment was given.

Physical Findings.—The patient is deeply jaundiced, well developed, muscular. Slight general superficial adenopathy. Heart and lungs are normal. No capillary pulse. Liver and spleen are not palpable. Right epigastric region is tender, especially beneath the right border of the rectus. Temperature is normal.

Laboratory Findings.—Blood-serum contains much bile-pigment and salts. R. B. C., 5,400,000; Hgb., 90 per cent.; W. B. C., 9400; fragility of red cells, .40 to .26. Wassermann tests negative with syphilitic liver and lipoid antigens, but strongly positive with cholesterinized antigen.

The urine contains a large amount of bile-pigment, bile salts and acids, urobilinogen, and a small quantity of urobilin. No albumin, sugar, or casts.

The stools contain no bile-pigment.

The duodenal content is bright yellow and contains no bile-pigment or acids. Spirochetes could not be demonstrated in smears.

Comment.—This is a case of complete jaundice (bile-pigment and bile acids present in large amount in both blood-serum and urine, but absent in the duodenal content). The urine gives a positive test for urobilinogen and urobilin, the explanation of which is not clear.

The etiology of this case is not clear. The fact that the Wassermann test (except with cholesterinized antigen) is negative in the presence of jaundice speaks rather against a syphilitic origin. It may be one of the epidemic type of infections. The patient will be treated with neosalvarsan on the assumption that arsenic is beneficial in all spirochete infections.

Diagnosis.—"Catarrhal" jaundice.

Hematogenous jaundice (acholemic, acholuric) is due to the retention in the blood-stream of the débris of hemolysis insufficiently transformed or eliminated by the liver cells. So much bile-pigment may thus be formed that the liver is fairly inundated, although great quantities of pigment may be eliminated (chocolate-colored duodenal content, pleochromia). The amount of pigment thus liberated in a comparatively short period of time may be enormous. The net clinical result of such massive hemolysis is the production of intense anemia without toxic phenomena (acholemia). The bile salts and toxic substances do not appear in the blood-stream or urine. While most cases of chronic hematogenous jaundice present but mild icterus, which is little more than a cosmetic defect, much more severe manifestations may be present (acholuric crises).

Illustrative Case.—The *history* of this patient is of great interest. He is fifty-five years of age. Since the age of fourteen he has been jaundiced. At that time there occurred a febrile attack associated with jaundice. At twenty years he had chills and fever with jaundice, which remained deep for two years, and never entirely disappeared up to the time of our first observation. For twenty-one years he was never entirely free from jaundice.

Febrile attacks, with intensification of icterus, occurred about once a month, and later about once in two weeks. In later years he noticed a prominence in the splenic region especially during attacks.

In January, 1914, a typical gall-stone attack occurred (colic and fever). After a month of fever and pain a gangrenous gall-bladder containing four pigment stones was removed at operation.

The persistence of crises of pain and fever brought the patient under our observation in 1915. Jaundice has been present in the family through four generations. Six of seven brothers and sisters were icteric. Two of three children of the patient have been chronically jaundiced.

In 1915 the patient was anemic and skin was lemon-tinged,

although there was no evidence of cholemia. The urine contained urobilin and urobilinogen. The spleen, firm and painless, extended about 20 cm. below the costal margin. The liver was palpable two fingerbreadths below the rib edge.

Red blood-cells averaged 4,000,000; Hgb., 70 per cent.; W. B. C., 4500; fragility of red cells, .52 to .36.

The stained smear resembled that of secondary anemia.

Wassermann tests were negative.

During an acholuric crisis the duodenal content, of dark mahogany-brown color, contained a great quantity of bile-pigment.

A diagnosis of chronic hemolytic icterus of family type was made. In April, 1915, the spleen was removed. Urobilin disappeared from the urine. Jaundice disappeared after a week. The skin was clear for the first time in twenty-one years.

Since operation the patient has had six attacks of pain in the liver region, with jaundice five times, never lasting longer than three weeks. These attacks occurred fifteen days, forty-five days, seven, nine, sixteen, and twenty months after operation. Liver has been tender after these attacks.

At the present time the scleræ are subicteric and the skin is dark. The patient has gained 25 pounds since operation. He feels distinctly better than he has felt in years. The splenic area is soft and pliable. Liver dulness is larger than normal. The slightly tender edge is palpable two fingerbreadths below the rib.

Blood-serum contains no bile-pigment, salts, or urobilin. Red cell fragility, .50 to .26; R. B. C., 5,400,000; Hgb., 90 per cent. (Dare); W. B. C., 12,000.

The urine contains no bile-pigment, bile salts, urobilin, albumin, sugar, or casts.

The duodenal content is of clear lemon color, a marked contrast to the former chocolate color. Bile-pigment and salts are present.

Diagnosis.—Chronic hemolytic icterus—family type. Acholuric and choluric crises.

Comment.—In presenting this case at this time, twenty-three months following splenectomy, it seems safe to state that there has been a complete recovery as far as the chronic hemolytic icterus is concerned. There is no evidence of hemolytic icterus at present, although the patient has had two mild attacks of hepatogenous jaundice during the twenty-three months since operation. These attacks may be attributed to dense adhesions about the gall-bladder noted at both operations. The patient is not now anemic. He enjoys good health, the first since his early manhood.

The icteric appearance of the subconjunctival tissues may be explained on the basis of vital tissue staining during the many years of jaundice. There is no other evidence of jaundice present, *i. e.*, the urine, blood-serum, and duodenal content give normal reactions.

Dissociation Jaundice.—The idea expressed in the term "dissociation jaundice" includes the conception that the blood-serum may retain the pigment, giving it up neither to the tissues nor to the urine, that the liver may shunt either the pigment or salts into the blood-stream independently, and that the renal filter may excrete the pigment or bile salts independently (Hoover and Blankenhorn). This is but a convenient way of expressing some of the physiologic phenomena observed in jaundice.

Illustrative Case.—The patient, Mrs. L., is a Mexican woman, forty-three years of age. She complains of attacks of great weakness and anorexia with remissions. Four years ago, in Cuba, the present symptoms began and lasted for nine months. She then felt well for nearly a year, after which the previous symptoms returned. She vomited daily. A blood transfusion was performed in the County Hospital. She would not submit to splenectomy.

When eight years old she had small-pox; at nine years, typhoid. Tonsillitis attacks were frequent. There is no family history of similar illness. A child born four years ago died at thirteen months. One miscarriage in 1913.

Upon examination the brown pigmented skin and the poorly

developed body are noted. The scleræ are muddy, but not definitely pigmented. The report of ophthalmoscopic examination states the presence of extensive retinitis of both eyes. There are no alveolar abscesses present (radiographs). Tonsils are large. The heart is slightly enlarged to the left. A systolic blow is heard at the apex and over the base. Lungs are apparently normal. Spleen edge is palpable, hard. Liver is not palpable. Wassermann test is negative.

The blood-picture is characteristic of pernicious anemia except for the absence of nucleated red cells in repeated examinations. R. B. C., 2,080,000; Hgb., 55 per cent. (Dare); W. B. C., 4000.

No bile-pigments or salts could be demonstrated in blood-serum or urine. The urine is normal except for large amounts of urobilin.

Diagnosis.—Pernicious anemia.

Comment.—This patient is presented in order to emphasize the icteric feature of pernicious anemia. With the tremendous blood destruction that occurs there appear in the urine great quantities of urobilin. This is an expression of hemolysis.

On account of the nationality of the patient (Mexican) it is difficult to determine whether she appears jaundiced. Aside from nationality and individual peculiarities one may have great difficulty in deciding whether a pernicious anemia patient is jaundiced. The subconjunctival tissues appear yellow, and there is a lemon tinge to the skin, without demonstrable jaundice in most cases of pernicious anemia. Yet occasionally one can demonstrate jaundice in these cases. The icterus is of the hemolytic type.

CLINIC OF DR. FRANK SMITHIES

AUGUSTANA HOSPITAL

CASES OF CHRONIC ENTEROCOLITIS ASSOCIATED WITH THE PRESENCE OF PROTOZOA IN THE STOOLS

(*a*) **A Cachectic, Toxic Young Female with the Complaint of Obstinate Constipation.**

(*b*) **An Anemic, Feeble, Adult Male with So-called "Achylia Gastrica" and Persistent Diarrhea.**

(*c*) **A Robust Male with Intermittent, Prostrating Diarrhea and Gastric Hyperacidity.**

CLINICAL CONSIDERATION OF PROTOZOIC ENTEROCOLITIS IN THE MIDDLE WEST

Sources of Stool Contamination; Etiologic Factors; Symptoms and Signs; Methods for Examining Stools Suspected of Harboring Protozoa; the Grouping of Protozoa; Incidence of Chronic Amebiasis, Trichomoniasis, Cercomoniasis, etc.; Protozoa Carriers.

TREATMENT OF PROTOZOIC ENTEROCOLITIS IN DETAIL

THE 3 patients which I present are of interest chiefly because, although they exhibit a rather wide diversity of clinical phenomena, they are similar in at least one respect, namely, in each case there is a chronic enterocolitis and in the freshly passed stools are found protozoa.

A decade since, the finding of protozoa (entamebæ, cercomonads, trichomonads, lamblia, etc.) in the stools of patients residing in the temperate zone was considered either as a curiosity or an unrelated accidental infection. An explanation that might be offered is that only within the past few years have

the stools of patients residing in this section of the country been systematically and properly examined for intestinal parasites of the protozoic group. The acquisition of the Philippine Islands, the possession of certain islands in the West Indies, and the garrisoning of the Mexican frontier and Panama have removed the American people from relative isolation with respect to these intestinal infections. At present there is a constant stream of travelers between all parts of the United States to the island possessions, Panama, the Mexican frontier, and Central and South America. While away from home many such people have classical attacks of dysentery, recover after treatment for the immediate illness, and become factors of no little etiologic significance with regard to spreading infection in their native communities. Other travelers do not have typical dysentery while away from home, but, as has been pointed out by Willets, they have their intestinal canals definitely infected with protozoa without untoward symptoms, and later become either sources of contamination or sufferers from enterocolitis.

The point I wish to emphasize is that in the Middle West at least there are numerous instances of enterocolitis associated with protozoa. These protozoa are in every way similar to those which inhabit the intestinal canals of individuals residing in tropical countries—in fact, many of the strains of protozoa have undoubtedly been imported from the tropics. The cases which I am about to detail illustrate the clinical aspects of protozoa carriers in the Middle West. It will be readily noted that rarely do these protozoa produce those violent clinical upsets for which they are frequently responsible in the tropics. It would seem that the change in habitat may have produced alterations in the virulence of these protozoa, so that resulting clinical upsets are of a milder character than those observed in southern latitudes. Instead of acute, often fatal, cholera-form enterocolitis, *it would seem that we must recognize a chronic enterocolitis associated with protozoa of generally low virulence, which virulence is, however, subject to fluctuations, with the consequence that more or less acute exacerbations*

of the disease may occur. It cannot be stated that between the acute exacerbations the parasitic infection is wholly innocuous.

CASE I.—A CACHECTIC, TOXIC YOUNG FEMALE WITH THE COMPLAINT OF OBSTINATE CONSTIPATION.

Mrs. L., Polish housewife, age thirty, born and raised in Michigan near St. Joseph, and a resident of Chicago for three years.

Presenting Complaint.—Chronic "dyspepsia," characterized by distress in the region of the stomach, belching of gas, and occasionally sour fluid, obstinate constipation for the past ten years, occasional vomiting, marked loss of weight and strength.

Past History.—Was very well as a child, with the exception of an occasional attack of grippe. Menstruated at age of twelve, and was in perfect health when married nine years ago.

Habits, Etc.—Resided on a farm near St. Joseph, Michigan, until coming to Chicago three years since. The water supply was from a well located between the house and barn-yard, and at a lower level than either the house or barn. Has always eaten abundantly of home-grown garden truck, bananas, and other fresh fruits. Has taken such in large quantities since constipation began.

Has no children and has never been pregnant.

Present Trouble.—About eight years since became "dyspeptic" without apparent cause. Experienced sour belching soon after meals and some abdominal distress. The discomfort bore no relation to diet. Vomiting occasionally occurred, but retention vomitus was never noted. Became very constipated, and would go longer than a week without a movement unless cathartics were used. Catharsis seemed to relieve dyspeptic disturbances.

Appetite diminished, and during the past eight months the patient has lost 104 pounds. Appetite is now poor; patient tires easily upon slight exertion, sleeps poorly, and is inclined to be melancholic.

Physical Examination.—It will be seen that we have to deal with a tired looking, apathetic, much undernourished female

(Figs. 172, 173). The skin and mucous surfaces are pale, cool, and dry. There is a trace of edema about the ankles. Apart from the pallor there is nothing abnormal in the *sclerotics;* the *eye-grounds* are negative. There is a low-grade infection in the *tonsils.* The *thyroid* is atrophic. The *heart muscle sounds* are weak, but the valve tones are clear. The *lungs* exhibit no

Fig. 172.—Case I: Photograph made about ten months before patient came under observation. Weight, 204 pounds.

lesion. The *abdomen* is moderately distended, with bulging in the region of the navel. It feels distended like a partially inflated football on palpation. There is some muscular tenseness in the right lower quadrant upon deep pressure, but no well-defined area of tenderness. There is no grossly demonstrable anomaly of the liver, spleen, or kidneys. The *rectal*

examination discloses a tight sphincter, internal painful hemorrhoids, and a rectum that has a boggy feel, such as is common when very frequent enemata have been given. The *uterus* is in good position and of normal size. The *reflexes* are normally active.

Laboratory Examinations.—*Test-meal.*—No stagnation or hypersecretion. Free HCl, 47; total acidity, 68. Blood tests negative. Microscopically, yeasts are present in slight excess.

Fig. 173.—Case I: Photograph taken when patient entered hospital. Weight loss more than 100 pounds in about eight months. Observe evidences of emaciation, flaccidity of muscles, and distended abdomen. (Infection with cercomonads and Entameba histolytica.)

Blood Examination.—Hgb., 70 per cent.; R. B. C., 4,200,000; W. B. C., 6600. The stained smear reveals no atypic red cells. The differential count is without interest other than the presence of 7 per cent. eosinophils. *Serologic tests* are negative.

Urine Analysis.—Twenty-four-hour quantity, 15 ounces;

specific gravity, 1012. Reaction, alkaline to litmus. Chemic and microscopic examinations demonstrate no abnormality.

Stool.—Specimen collected after patient had been on very soft diet for thirty-six hours and 2 ounces of solution of magnesium sulphate had been administered. The third stool passed was examined immediately after its being voided. *Grossly*, the specimen was a thin purée, chocolate-brown in color, somewhat foamy, and had a peculiar "fleshy" or fruity odor. There was very little food residue and no excess of mucus. The *chemical examination* indicated rather pronounced *alkalinity* to litmus and to phenolphthalein; benzidin test for *altered blood* was faintly positive (patient had been meat and meat-product free for forty hours); *hydrobilirubin* was present (Schmidt bichlorid test); pancreatic functional test revealed trypsin less than 500 units (low) and amylase between 2000 to 3000 units (within normal range).

Microscopic Examination.—From three to as many as nine very actively motile cercomonads to a high-power field; many encysted entamebæ of the histolytica type, and an occasional sluggishly motile form; great numbers of cocci, many in short chains; yeasts, long motile and non-motile bacilli, and a few very active spirillæ. Many triple phosphate crystals.

Röntgen Examination.—A low *stomach*, with active peristalsis and normal contour. The pylorus was open and the duodenal contour was normal. The duodenum emptied rapidly. Both *duodenum* and stomach were readily displaced upon palpation. There were no areas of tenderness. The *colon* was moderately distended throughout, without any evidence of obstruction; haustral contractions were few and incomplete; there was a tendency to "puddling" in the cecum and in the redundant sigmoid loop.

Proctoscopic Examination (after preparation by administering 2 ounces of castor oil and giving two colon flushings with warm soapy water).—The mucous membrane was pale and moderately edematous; there were several easily traumatized hemorrhoidal swellings just inside the sphincter. Otherwise the examination was negative.

Remarks.—The patient was considered as an instance of malnourishment due to chronic enterocolitis. It was deemed that the infection of the terminal ileum and the colon with protozoa could not be disregarded as a cause of the disability. Accordingly, after hemorrhoidectomy and measures directed toward clearing up the infections in the mouth and throat, the patient was put to bed and treatment to rid the gut of parasites was instituted (see below). Improvement was slow, but the patient gained 28 pounds in weight and was able to resume her household duties in seven weeks. The bowel was free from protozoa after three weeks' treatment in the hospital.

CASE II.—AN ANEMIC, FEEBLE ADULT MALE, WITH SO-CALLED "ACHYLIA GASTRICA" AND PERSISTENT DIARRHEA.

Mr. S. H. A., a married male, subtreasury clerk, aged fifty-five, comes to the hospital on account of "rumbling and miserable feeling in the bowels," loss of strength, weight, and appetite, headaches, diarrhea, eructations of food and gas, and malaise. Has been treated for "achylia gastrica" and "intestinal indigestion" for several years without relief.

Past History.—Common infectious ailments of childhood. Had "malaria" (chills and fever for which he took quinin) twenty years ago. Has never lived in the South, and never out of the latitude of Illinois except for a trip to Los Angeles, California, in an attempt to regain health, about two years ago.

Habits.—Denies venereal disease; has lived regularly, but worked hard and under mental stress for twenty-five years; smokes two or three mild cigars daily; uses no alcoholics or stimulants; eats rapidly and has been fond of lettuce, radishes, celery, and fresh fruits all his life, and has eaten of them very liberally. Is the father of three children and his wife never miscarried.

Present ailment manifested itself about six years ago. He began to be distressed with "*rumbling*" and *discomfort in the abdomen.* The trouble commonly appeared soon after eating and was not relieved by various forms of diet. The rumbling was accompanied by discomfort, but *no actual pain*—the sensa-

tion was as though he had taken a cathartic. The rumbling was often accompanied by noises so audible as to be embarrassing. Very little gas was passed and there was no diarrhea, although the *stools were generally soft and of foul odor.* Took treatment from a physician, but received no benefit. Two years since was told that he had "*achylia gastrica.*" It was also feared that he might be developing cancer somewhere in his gastro-intestinal tract or that "pernicious anemia" threatened. Had his *stomach function tested,* but *stools were only examined for "occult blood."* He was told that there was no acid secretion in his stomach, but that cancer was absent because the test for blood in the stools was negative. Shortly afterward became troubled with *eructations of "gas" and food* soon after meals; had no distress in this regard between meals except at night, when his mouth would fill "with sweet water." Frequently experienced *nausea, but never vomited. Headaches* toward evening became annoying, and *dizziness* and *weakness* with *weight loss* gradually supervened. *A year ago diarrhea stools became constant.* Usually two to four very soft or watery stools are passed before 10 A. M. There is no pain. The stools have recently been *copious, very foamy, and of such disagreeable odor* as to "scent the whole house." They have commonly been light colored, never clay colored nor of fatty appearance. The *appetite* has become very capricious—nothing tastes "right." Has become *short of breath; heart palpitates on slight exertion;* is now very nervous and apprehensive, because he fears that he may be affected with a malignant, incurable ailment.

Physical Examination.—You will observe that this patient is thin, pale, sallow, and tired looking. The anemia, undernourishment, and weakness when considered with respect to his age (fifty-five years) readily suggest the possibilities of malignancy or a grave anemia. The patient has, however, lost but 5 pounds during the past year and only 22 pounds during the past five years. It is unusual for a cancer patient to lose weight so gradually, and it is likewise unusual for cancer to persist for as long as five years without a fatal termination. The *skin* is pale and sallow, without having the lemon or canary yellow cast

to it, as is common in instances of "primary" anemia. It is dry and cool, and not moist, "greasy," and warm as in "pernicious" anemia. There is no *peripheral edema*. The *sclerotics* are pale, but not tinged with yellow. The *tonsils* are moderately enlarged, but free from crypts. The *teeth* are well repaired. The *lungs* are normal. The *heart sounds* are weak, but clear. The systolic *blood-pressure* is 120, and the diastolic 82.

The *abdomen* is well rounded, in rather sharp contrast to the sunken, "scaphoid" abdomen common to malignant disease. You will observe rapidly moving, *undulating, peristaltic waves in the region of the navel.* They are apparently painless and are aggravated upon my tapping the abdomen. When the belly is tapped the movements of the gut produce audible gurgles. The position of the peristaltic waves—namely, in the region of the navel and passing generally toward the right iliac fossa—would appear to indicate that the visible movements represent exaggerated rate of contraction of the small bowel, particularly the ileum. Palpation demonstrates a thin abdominal wall, through which gas-distended loops of gut can readily be felt as they fill and empty. The colon is dilated, especially its right third. There is some distress on moderate pressure over the cecum.

Rectal examination is negative, with the exception of large, irritated hemorrhoidal tags. The prostate is diffusely enlarged, but smooth.

Proctoscopic examination reveals an edematous, pale mucosa, stippled with many superficial erosions, but no definite ulcers. There is rather profuse bleeding as the sigmoidoscope is passed. The sigmoid is free from ulceration.

Ophthalmoscopic Examination.—Veins moderately engorged. Disk negative.

Reflexes.—Nothing abnormal.

Laboratory Examinations.—*Test-meal.*—Stomach empties rather rapidly. Free HCl, 0; total acidity, 22. Mucus in moderate excess. No blood. Microscopically, few yeasts; great numbers of diplococci and short chains of streptococci.

Blood Examination.—Hgb., 60 per cent.; R. B. C., 3,740,000; W. B. C., 5800. Stained smear reveals no abnormal red cells.

Differential Count.—Small lymphocytes, 27 per cent.; large mononuclears, 8 per cent.; transitionals, 2 per cent.; polynuclears, 62 per cent.; eosinophils, 2 per cent.

Wassermann Reaction.—Negative.

Röntgen Examination.—Esophagus normal. Stomach rather large and low. Tone is good, the peristalsis being vigorous. There is no abnormality in either gastric or *duodenal* contour, and no stagnation. On palpation, there are somewhat tender areas over the gall-bladder area and in the right iliac fossa.

Colon plate reveals no abnormality, with the exception of a large, gas-distended cecum and ascending colon and a rather noticeable reflux of cecal contents into the terminal ileum.

Stool Examination.—After free catharsis with a saline laxative the first three stools collected and examined warm revealed vegetable cellular waste, few poorly broken up meat fibers, and great numbers of cocci, actively motile small and large rods, torulæ, and few spirillæ. No parasites were seen in the careful examination of eighteen smears. The very abundant and active bacterial flora suggested liquid diet for a day, the administration of another saline cathartic, and the collection of more stools. The second stool passed after this régime contained enormous numbers of large, very motile *trichomonads* and few *cercomonads*, as well as many spirillæ, moving rapidly with eel-like motions. There were groups of leucin crystals, few cholesterin plates, and many fat needles. This stool was definitely alkaline and contained blood-pigment when tested with benzidin. *Tryptic activity* was much reduced, but *amylolytic* power was greater than 1000 units. *Hydrobilirubin* was present.

Remarks.—This case has been treated not only as an instance of absent hydrochloric acid associated with chronic atrophic catarrhal gastritis, but has been attacked from the standpoint of the demonstrated intestinal infection (see below). There is already improvement; the rumbling has greatly diminished; the patient is regaining appetite and strength, and there

has been a slight gain in weight. The stools have been now free from parasites for nearly a week (after ten days' therapy).

NOTE.—At the end of a month's hospital incarceration the patient was discharged.

There had been a definite gain in weight, strength, and blood volume and quality. After five months the patient returned for observation. He has been working constantly for nearly four months. His stools were parasite free; the Hgb., 90 per cent.; the R. B. C., 4,800,000; W. B. C., 9000, and the differential count showed but 1 per cent. of eosinophils and 77 per cent. polynuclears. There have been but two transient attacks of diarrheic stools. Appetite and taste are excellent; he is on a fairly full diet, with limitation of proteins, and has gained 17 pounds. "Rumbling" of the bowels is rare; when it is experienced it is commonly in the morning, and the patient thinks it caused by a too heavy breakfast and the smoking of a cigar as he drives to his office.

CASE III.—A ROBUST MALE WITH INTERMITTENT, PROSTRATING DIARRHEA AND GASTRIC HYPERACIDITY.

This patient is exhibited two and one-half years after treatment for enterocolitis associated with protozoa in order to emphasize the facts that protozoa may be found in the stools of patients affected with gastric hyperacidity; that treating the gut ailment from the standpoint of these infecting parasites results in alleviation of distress, and that, if properly supervised, such treatment apparently results in a permanent cure. A brief summary of this patient's history is of interest.

Nearly three years ago Mr. K., who was born and raised in one of the north-shore suburbs, sought relief for periodic, prostrating attacks of diarrhea. These attacks came on with especial severity in the Spring and the Fall. As many as thirty watery, foul-smelling, movements a day were experienced. The attacks came on quite irrespective of diet or care with respect to personal hygiene. In the attacks the patient would lose as much as 25 to 35 pounds in weight. He was suspected of having tuberculosis of the bowels. Headaches, loss

of appetite and strength accompanied the attacks of diarrhea. There was rarely abdominal pain.

Certain observations with regard to the patient's habits and mode of life are valuable. He is an ardent hunter and fisher. His late Summers and parts of the Falls are spent following the streams and living in the woods. He frequently is compelled to drink contaminated water. In the Springs and Summers he is engaged in cultivating flowers and raising garden truck for the market. From boyhood to his present age (forty-five) he has been an enormous eater of garden vegetables. His appetite for such is so keen that he rarely goes a day without raw green vegetables of some kind. He frequently eats lettuce hearts, radishes, celery, and young cabbage right from the field. It will be observed that it is usually in the Spring, when garden truck is particularly delicious, or late in the Fall, when contaminated water has been drunk, that the patient experiences his most pronounced attacks of diarrhea.

At the time of entrance to the hospital this patient was 26 pounds underweight, was toxic appearing, had hemoglobin of 70 per cent., and eosinophilia of 5 per cent.; had a gastric acidity of 86 free HCl and 98 total acidity, and was passing frequent watery stools. There was moderate abdominal distention and some tenderness over the gall-bladder region. Proctoscopic examination disclosed many superficial ulcers of the upper rectum and the lower sigmoid. Stools examined even without catharsis contained great numbers of actively motile trichomonads and entamebæ of the histolytica type, together with a rich bacterial flora and many triple phosphate crystals. Blood was promptly demonstrated by the benzidin test. The urine was negative.

The patient was put to bed on ipecac, calomel, and thymol treatment, with daily colon lavages with thymol 1 : 5000 and quinin 1 : 3000. The trichomonads disappeared rapidly from the stools, but before the entamebæ were lost it was necessary to administer large doses of thymol by mouth and give a kerosene enema. The patient was discharged from the hospital in about three weeks, with instructions to avoid contaminated

water and foods. He has reappeared for examination twice in two and one-half years and each time the stools have been parasite free.

The improvement in his general health has been marked. You will observe that he appears far from an invalid. His weight is close to 200 pounds; he has no headaches; is diarrhea free, and his hemoglobin is practically 100 per cent. (Tallqvist).

DISCUSSION

From the brief review of the above cases selected from instances of enterocolitis associated with protozoa, it would seem that a summary of instances observed in my clinic may be of service.

Study of the records of the last 1000 stool analyses in my clinic at Augustana Hospital indicates that in 93 instances protozoa were observed. The patients furnishing these specimens were largely from the northern half of the Middle West —a region where man is commonly considered to be free from protozoic infection.

Apart from those in tropical countries, but little mention of such findings exists in medical text-books. Vague, archaic descriptions of the parasites, together with illustrations common to laboratory manuals for decades, have been firmly engrafted on the medical mind throughout the country—and this, in spite of the emphatic and complete articles by Darling, Craig, Deeks, Freund, Sistrunk, Lyons, and Giffin. It would seem quite possible that the local and general increase in population and the ease with which people get from place to place is worth emphasis. As I have mentioned, the free intercourse between Mexico, South America, the Islands of the far East, and the Southern States of this country may be a not to be neglected factor in a wider spreading of protozoic intestinal infections than has heretofore been recognized.

Etiologic Factors of the Group.—The patients' *ages* ranged from seven to eighty-two years. The average age was thirty-nine.

Sex.—There were 51 males and 42 females. There was practically no difference noted in the sex-age figure.

Nativity.—Fifty-two patients were of Scandinavian birth or extraction. The remainder were Americans, Germans, Irish, Russians, Austrians, or English.

Geographic Distribution.—During the past ten years we have had strongly impressed upon us the importance of gaining information when taking histories regarding both the present and past residences of patients, and also the exact environmental conditions under which they have lived. These points appear to have especial significance in the examination of individuals affected with obscure abdominal ailments, particularly when such are associated with diarrhea.

The geographic diversity of patients forming this group was as follows: Illinois, 29; Iowa, 16; Wisconsin, 13; Nebraska, 8; Michigan, 7; Minnesota, 6; Indiana, 4; S. Dakota, 2; Arkansas, 2; Ohio, 2; Texas, 2; Kentucky, 1; N. California, 1.

It will be noted that but 4 patients came from regions south of Illinois. There were 11 patients who had made occasional business or pleasure trips to the Southern States. There were 78 individuals who had never been south of Illinois.

Water-supply, Etc.—We routinely seek information not only with respect to the patient's present drinking-water but also regarding his drinking-water from childhood. The importance of water-supply as a source of protozoic infection has been abundantly commented upon in the literature; clinically, we have gained valuable facts from seeking information about it. Not rarely patients admit that for years their drinking-water has been bad, especially such patients as have taken up residence in recently opened "homestead" territory. Shallow, dug wells, contaminated springs, sluggish, weed-grown rivers or ponds, or lakes befouled by drainage systems have proved to be the source of water-supply in 78 instances. In the remaining 15 cases the drinking-water was seemingly good.

TABLE I

CASES OF PROTOZOIC ENTEROCOLITIS IN THREE YEARS

Total Number, 93

Patients with intermittent or chronic diarrhea	86
Patients without diarrhea	7
Patients from south of Springfield, Illinois	4
Patients who had visited in the South	11
Patients who had never been in the South	78
Patients from small towns, rural communities, or farms	70
City or large-town dwellers	23
Patients who had habitually drunken well or shallow river water	78
Patients whose water-supply was seemingly good	15
Patients who were large eaters of fresh garden truck, bananas, or unwashed fruits	66

The contamination of garden vegetables by dung is not unlikely responsible for protozoic spread. It is well known that hogs, cattle, sheep, barn-yard fowls, or snails, worms, and flies are common carriers of flagellate protozoa and ameba in their intestinal canals. Instances where cholera-form epidemics have existed on stock farms are not unknown to veterinarians. It is the usual practice to "fertilize" fields or boxes in which head lettuce, radishes, tomatoes, mushrooms, celery, etc., are grown with "rich" manure. One cannot deny that those who partake largely of these vegetables, raw and often carelessly washed, are in danger of ingesting both active and spore forms of protozoa. Of late years truck-farming has become an important industry about every big city, hence the army of "manure-eaters" is not located wholly in the country: city folk who are financially able to afford the luxury of fresh garden truck may be contaminated during the entire year. The common house-fly not infrequently contaminates fruits, particularly the banana. This fact is well known to Mexican planters, who have cleared areas previously endemic with amebic dysentery by the simple procedure of screening the banana sheds and covering dung mounds with antiseptics, sand, or oil. It is quite possible that the prevalence of protozoa in the mouths of individuals in both

tropic and temperate countries is due to food and water contamination. These parasites live in filth; if the filth is supplied in the mouth by eroding gums and decaying, dirty teeth (as in "pyorrhea") the parasites may thrive and do harm. Soggy tonsils, cancerous, syphilitic, phagedenic, or tuberculous ulcers likewise furnish an ideal camping-ground for these unwelcome guests. If the hydrochloric acid of the stomach did not inhibit or destroy these parasites, it is not unlikely that intestinal infection would be more universal. What controlling effects digestive enzymes, bile salts, or intestinal flora have on these organisms is not known. The mechanical activity of the stomach, duodenum, and jejunum may prevent lodgment and multiplication in these parts of the gut, while the stagnation and increased bacterial proliferation of the terminal ileum and of the large bowel may furnish from time to time the proper stimulus to protozoic lodgment and growth. That protozoa may exist for a long time without causing symptoms is evidenced by Willets' report that 85 per cent. of all Filipinos habitually harbor these parasites, and that but intermittently (Spring and Summer) do the parasites enormously multiply and produce local and systemic effects.

Of our 93 cases, 66 patients were consistent eaters of fresh garden truck, unwashed fresh raw fruits, or bananas.

Clinical Symptomatology.—(1) *Diarrhea.*—In our series this was a presenting complaint in 86 cases. Diarrheic stools without frequent evacuations were not rarely passed chronically, with periods when the *movements were frequent.* The *intermittency of frequent diarrheic stools, with apparently normal motions in the intervals, is quite characteristic of this affection,* and aids in differentiating it from the diarrheic stools associated with gastric or pancreatic achylia, imperfect liver function, tuberculous or luetic ulceration of the bowel, and the like, in which ailments the *constancy* of the soft or fluid stools is a common observation. In the event of the diarrheas associated with protozoa the patient may note no change in his habit, mode of life, or diet, but quite suddenly may experience frequent evacuations of the bowels. Occasionally a period of

severe mental or physical overstrain or an attack of la grippe, tonsillitis, or the like are stated as preceding the diarrhea. The attacks may be infrequent or may come close together over a period of many years. The latter event may supervene upon the former. In our series one patient had had but three attacks, while another case had experienced nine in one year. There were 14 patients in whom the diarrhea had been continuous.

TABLE II

DIARRHEA PATIENTS

Duration:	
Less than one year	8
Between one and three years	27
Between three and five years	31
Between five and ten years	12
More than ten years	8
Total cases	86
Attacks of Diarrhea:	
Least number	3 in all
Maximum number	9 yearly

(Summary of 72 cases)

Diarrhea, Chronic and *Continuous* ..... 14 cases

The *duration* of the diarrhea varies greatly (Table II)—67 per cent. of our cases had been affected from one to five years; 8 cases had been ill less than one year, while a like number had been ailing for more than ten years, the longest period being forty-three years.

In *character* the diarrhea is commonly of the gassy type, without discomfort upon the passage of the stool. Occasionally an associated inflammatory change in the colon, especially in the sigmoid or rectal sections, results in painful evacuations. Hemorrhoids were present in 38 per cent. of our cases. They may become extremely annoying when the motions are very frequent and watery. The bowel movements are generally easy, but prolonged diarrhea may lead to marked tenesmus and prostration.

Constipation occurred in 4 of our cases; in fact, the most

toxic patient appeared for the relief of an obstinate intestinal stasis. Routine examination of the freshly passed stool following saline catharsis disclosed enormous numbers of entamebæ in the histolytica stage and actively motile cercomonads.

TABLE III

SUMMARY OF CLINICAL SIGNS AND SYMPTOMS

(Figures Stand for Number of Cases)

Clinical Observation.	Parasite. Ameba.	Cerco-monas.	Tricho-monas.	Lamblia.	Mega-stoma.	Balan-tidium.
Diarrhea	24	33	20	5	2	2
Constipation	1	2	1	0	0	0
Normal stools	2	1	0	0	0	0
Abdominal pain	21	30	17	4	2	1
Dyspepsia	15	28	21	5	1	2
Weight loss	17	30	21	3	2	2
Achylia gastrica	13	12	11	2	1	1
Subnormal acid	8	14	8	2	0	1
Normal gastric acid	6	10	2	1	1	0
Anemia	26	31	18	4	2	2
Eosinophilia (above 3 per cent.)	19	27	15	2	1	1
Blood in stools	14	17	16	3	2	2
Fever	4	1	2	0	0	0
Chill	2	3	2	1	0	1

(2) *Dyspepsia.*—This was a prominent complaint in 73 cases (75 per cent.). The dyspepsia was generally associated with nausea (particularly in the morning), eructations of foul gas, regurgitation of sour food mixtures (even when test-meal analyses revealed absent or low gastric acid), gassy distention of the abdomen, soreness after eating, loss of appetite, and occasionally vomiting.

(3) *Abdominal pain* or *discomfort* was a complaint in 89 instances (95.6 per cent.). The discomfort was variously described as colicky (like gas-pains following "abdominal exploration"), soreness, distention, cramp-like, "dragging," or vague "weak feelings." Sometimes the rapid peristaltic movements of the small gut were productive of an annoying discomfort, even though actual pain were absent.

(4) *Weight Loss.*—This was noted in 75 cases (80 per cent.). The loss varied from 5 to 104 pounds. The average loss was 17.3 pounds. Some weight losses were so striking and so rapid as to bring the patient under observation for suspected, atypically located malignancy: a young woman affected with entamebæ and cercomonads lost 104 pounds in less than one year; a middle-aged woman lost 80 pounds in less than fifteen months; a man, aged eighty-two, lost 65 pounds in less than ten months. With the exception of the first case, these patients nearly doubled their weights during the year following treatment. The first mentioned female gained 28 pounds within three months of her discharge from the hospital.

(5) *Strength loss* is often striking, even though the weight may have decreased comparatively little. Apart from the possible toxic effect of the protozoic infection, loss of sleep due to diarrhea, imperfect assimilation or diminished quantity of ingested food, continuous abdominal discomfort, anemia, and the dread of the existence of an incurable ailment seem to be important factors in the production of a general, systemic weakness.

(6) *Anemia* is usually evident, even though in some cases it may not be pronounced. It is sometimes of a grave degree; certain patients are sent in as pernicious anemia cases or as atypic anemias of unknown etiology.

(7) *Mental Attitude.*—A peculiar malaise or melancholia is frequently observed. It may be due to a dread of impending evil, continuous headaches (especially common in some instances), weakness, anemia, loss of sleep, abdominal distress, or toxic substances occurring as a consequence of the bowel upset.

Physical Examination.—(1) The patients generally appear both starved and toxic; in this respect the individuals past middle age closely resemble those with malignant disease. The skin often exhibits a dusky, sallow pallor. It is not rarely dry and wrinkled. Subcutaneous fat is lost quite early, although not so early as in malignant disease. There is apt to be edema about the ankles in the severe cases. The visible mucosæ are generally pale, dry, and scaly. The saliva is often scant and

tenacious. The tongue is usually swollen, marked by the teeth, and heavily coated. The breath is heavy and often foul. Focal infections about the nasal passages, teeth, and tonsils are not uncommon, but such are not characteristic for this affection.

Thoracic examination often discloses shallow respirations of increased rate, and when weight and strength loss have been rapid, such are associated with anemia and signs of lung edema. The heart-rate may be slowed, but upon exertion it increases out of reasonable proportion to the amount of exercise. Murmurs, generally systolic, are not uncommon in such cases.

Abdominal inspection revealed visible peristalsis over the small bowels in 22 of our patients (23 per cent.). The waves were often strikingly visible and of very rapid rate. In several instances peristalsis was seen only following sharp tapping of the belly wall with the fingers. Patients had sometimes felt the actively moving small guts and were under the impression that snakes, tapeworms, etc., had developed within them.

Palpation usually made out both small and large bowels filled with gas and feces. This was a common observation in the diarrhea cases. Distention of the terminal ileum was frequently pronounced—the coils felt as large as loops of normal colon. *Tenderness* over the small and large gut was noted in 64 cases (69 per cent.). In severe diarrhea it was often marked. Tender areas over the gall-bladder or appendix zones were present in 83 instances (88 per cent.). The liver was enlarged and tender in 3 patients. The spleen was palpable 9 times: in 4 cases there was definite enlargement.

Rectal Examination.—The frequency of hemorrhoids has already been commented upon. *Proctoscopic examinations* were made in the last 37 cases coming under observation. Bleeding ulcers were noted in the sigmoid or upper rectum 9 times. In 21 cases a diffuse catarrhal or granular inflammation of the gut was seemingly of greater grade than the frequent bowel evacuations alone would explain. In these cases the bowel was easily traumatized, and in some instances the bleeding following instrumentation was rather severe.

Laboratory Examinations. (1) *Stools.* For the detection of protozoa it is absolutely essential that the stool be properly collected. Our procedure is as follows: Any medicines which the patient may have been taking are stopped. He is then placed upon a liquid diet for thirty-six hours. At the expiration of this period he comes to the clinic. He is given 1 ounce of Epsom salts or 500 c.c. solution of citrate of magnesia upon an empty stomach. The first stool passed is discarded; the

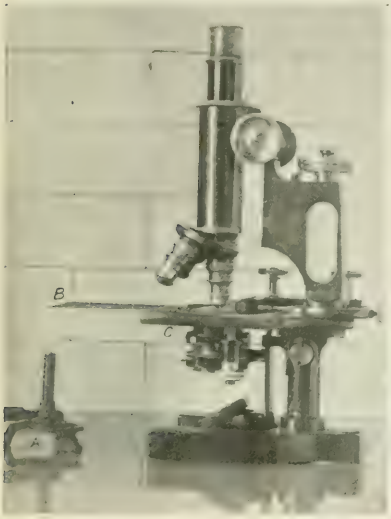


Fig. 174.—Practical improvised warm stage for use in examining stools for protozoa. Bunsen microburner (*A*) heating copper plate (*B*), fitting microscope stage (*C*). Copper plate is perforated above Abbé condenser.

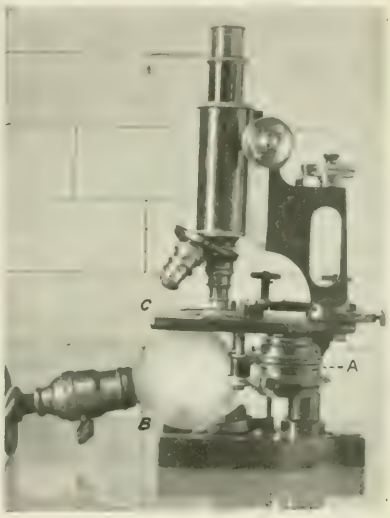


Fig. 175.—Simple arrangement for keeping specimens of stool warm while examining for protozoa. Abbé condenser (*A*) swung to side and carbon filament lamp (*B*) placed directly beneath microscope stage (*C*).

second or subsequent stools are collected in a warmed container, mixed with one-fourth volume of warm normal saline solution and examined at once, by high power, in thin smears placed upon warmed glass slides on the hot stage of a microscope. None of the ready-made warm stages are satisfactory. After trying all forms we have come back to the copper plate heated by a Bunsen microburner placed upon the ordinary microscope stage (Fig. 174). If such is not at hand, an old-time filament

electric bulb, placed beneath the microscope stage after the Abbé condenser has been swung to one side, answers very well (Fig. 175). In suspected cases examination is made of at least six slides before negative opinion is given. Very often examinations are made daily for even so long as a week where the history is suggestive. Frequent examinations of different stools often not only detect parasites that might have been missed, but lead to the discovery of multiple infections. High power should be used in the examinations, particularly for entamebæ, because the differentiation of the types may rest upon careful study of cellular architecture (Figs. 176

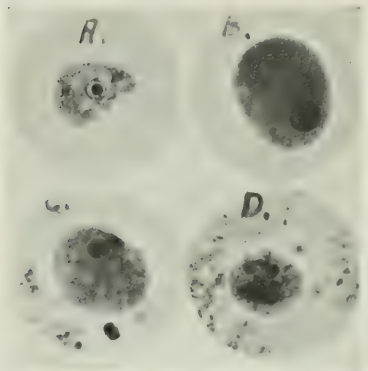


Fig. 176.—So-called "non-pathogenic" and "non-parasitic" types of entamebæ. *A*, Found in Manila water-supply and cultivated from the intestinal tract of man. *B*, *C*, *D*, Parasitic but "non-pathogenic" entamebæ found in the stools of "healthy" persons. *B*, Entameba coli; *C*, Entameba coli, variety niponica; *D*, Entameba coli, 8-nuclear cyst. (After Walker.)

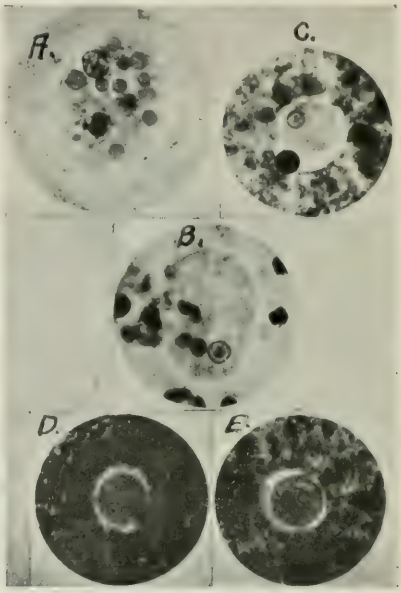


Fig. 177.—"Pathogenic" and "parasitic" entamebæ found in dysenteric stools. *A*, Entameba histolytica (Schaudinn); *B*, Entameba histolytica in tetragena stage; *C*, Entameba histolytica cyst; *D*, 1-nuclear stage preparatory to encystment; *E*, 2-nuclear stage. (After Walker.) Encystment occurs during recovery from disease. Persons harboring encysted entamebæ are carriers.

to 181). Stained specimens are best prepared by the Hastings-Giemsa method suggested by Darling.[1]

According to Willetts, protozoic infection in soldiers and inhabitants of the Philippines is very frequent. In his series entamebic infections were found in 37.5 per cent. of the 1000 individuals examined: in 38.8 per cent. of 900 Filipinos; in 26 per cent. of 100 Americans; in 46.3 per cent. of 417 adult female Filipinos; in 34.2 per cent. of 383 adult male Filipinos; in 25 per cent. of 100 Filipino children; in 25.4 per cent. of 71 adult male Americans; and in 27.6 per cent. of 29 adult female Americans. Double infections were found in 59.2 per cent.; pure *Entameba coli* infections in 35.5 per cent.; pure *E. histolytica* infections in 5.3 per cent.; *E. coli* infections in 94.7 per cent.; and *E. histolytica* infections in 64.5 per cent. If more stools had been examined of each patient it is believed that the positive percentage would have been about 55 instead of 37.5 per cent.

Strong, 1901, looking for *E. coli* in the stools of non-dysenteric persons of Manila, found 8 (or 4 per cent.) infected in 200 examinations. Vedder, 1906, examined 100 healthy individuals in the Cotabato Valley, Mindanao Province, for the presence of *E. coli*. Of 50 native scouts, mostly Moros, 35 (or 70 per cent.), and of 50 American soldiers, 25 (or 50 per cent.) were

[1] Fresh coverslip preparations containing a sufficient number of entamebæ to warrant staining and study, or those intended for diagnosis, are made into smears by sliding off the coverslip and thoroughly drying both slip and slide, after which each is stained with Hasting's stain for fifteen minutes. Satisfactory films are then overstained with Giemsa's stain until the film has a diffuse reddish-purple tint. The film is then plunged into 60 per cent. ethyl alcohol containing about 1 per cent. of water of ammonia (10 per cent.), and differentiated in this, washed in water, and controlled by the microscope until the purple substance of the nucleus and the blue color of the cytoplasm are strongly contrasted. The film when properly differentiated has a blue-violet color. If the film has been greatly overstained, it is treated with a momentary douche of 95 per cent. alcohol. Beautiful pictures are obtained in this way, but, what is of more importance, the various figures displayed by the purple staining substance (karyosome?) can be noted and followed with ease. This purple staining substance in the nucleus of *Entameba tetragena* in dried, fixed films represents only a portion of the nucleus, as the centriole and peripheral chromatin do not stain purple by the above method. The purple staining substance in the nucleus of *E. tetragena* frequently appears as a ring, or as a reticulum or scattered granules.

infected. Ashburn and Craig, 1907, examined 107 healthy American soldiers at the Division Hospital, Manila, for entamebæ. They found *E. coli* in 76 (or 71 per cent.) and *E. histolytica* in 4 (or 3.7 per cent.). Hoyt, 1908, reported 34.6 per cent. infected of 300 persons examined for active entamebæ at the Naval Hospital, Canacao, Cavite Province. Of 283 American sailors, 32 per cent., and of 17 Filipinos, 76 per cent., were

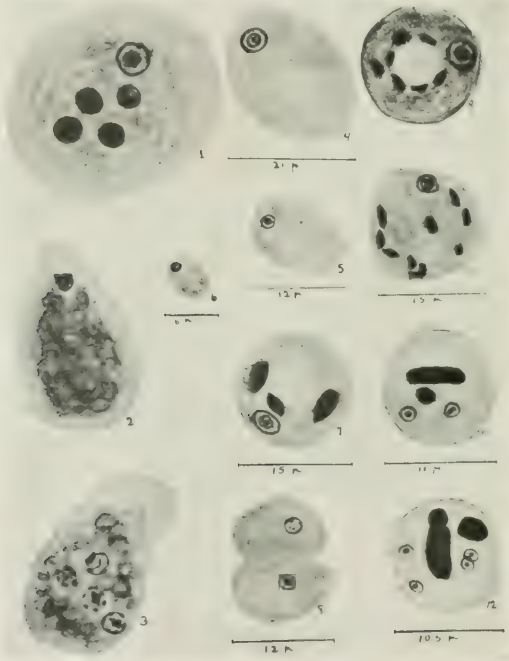


Fig. 178.—Encysted Entamebæ histolytica showing nuclear units and chromidial fragments. (After Darling.)

positive. Garrison, 1908, in examining 4106 inmates of Bilibid Prison, Manila, for general evidences of intestinal parasitism, found 23 per cent. infected with active entamebæ. In a similar study Garrison, Leynes, and Llamas, 1909, found 2.7 per cent. infected with active entamebæ among 1000 inhabitants of Taytay, Rizal Province. Rissler and Gomez, 1910, report 0.39 per cent. positive for entamebæ of 6018 persons examined at Las Pinas, Rizal Province, for evidence of intestinal parasit-

ism, particularly hookworm infection. In similar investigations the same men found not a single entamebic infection among 2549 persons examined at Tuguegarao, Cegayan Province, and 802 examined at Santa Isabel, Ilagan, Isabela Province. Stitt, 1911, examined 100 Filipinos of the outdoor clinic of the Naval Hospital, Canacao, Cavite Province, for entamebæ, finding 9 per cent. positive.

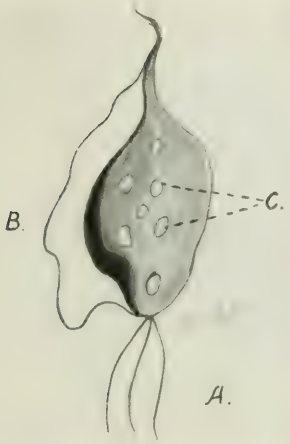


Fig. 179.—Trichomonas hominis intestinalis. Sketch previous to encystment, while sluggishly motile. *A*, Flagella; *B*, undulating membrane; *C*, vacuoles and nuclear particles.

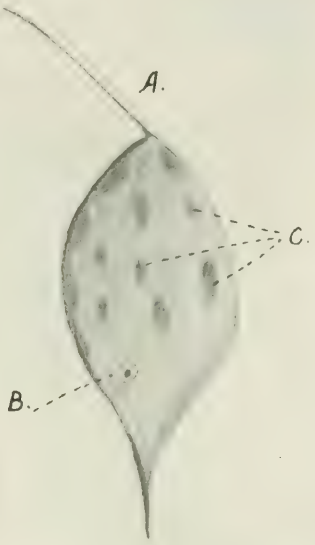


Fig. 180.—Cercomonas hominis intestinalis. Sketch at actively motile stage. *A*, Flagellum; *B*, nucleus; *C*, bacteria and food débris.

Walker has given the following description of the quiescent and encysted forms of entamebæ:

The resting entameba is distinguished from other bodies found in the stool by its size, distinctness, regularity of contour, degree of refractiveness, and especially by its nuclear structure. The entamebæ vary in size within considerable limits, but are usually from 20 to 30 microns in diameter. They are, therefore, larger than pus cells or other protozoa, with the

exception of *Balantidium coli* that are found in the stools of man. They are also more refractive than pus, epithelial, or other cells found in the stools. The nuclear structure of the entamebæ is particularly characteristic. The unencysted entameba possesses, unless in the process of division, only a single nucleus. This nucleus is round, or occasionally slightly oval or irregular, small with reference to the size of the cell, and appears not solid, but as a refractive ring. This relatively small, ring-shaped nucleus appears to be absolutely diagnostic of an entameba. Only one other kind of cell observed in stools possesses a nucleus in any way resembling that of an entameba.

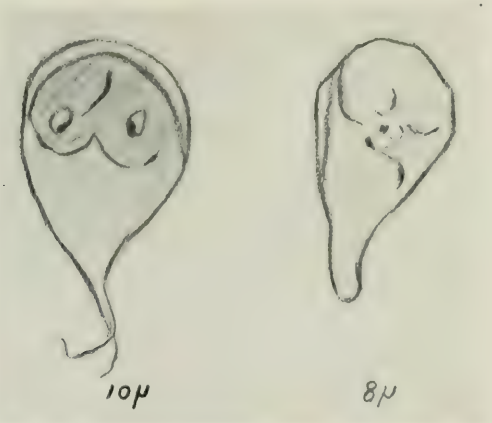


Fig. 181.—Lamblia intestinalis. Sketch from specimen of stool. (Flagella poorly indicated.)

This is an epithelioid cell, sometimes found in mucous stools, which has a ring-form nucleus relatively much larger than that of an entameba, occupying one-fourth to one-half of the cell. While an entameba may occasionally be observed with an abnormally large nucleus, probably preparatory to division, the nucleus never approaches the size of the nucleus of this epithelioid cell. The latter cells are also less refractive and granular than entamebæ.

The encysted entameba is round or slightly oval, more refractive than the resting or motile stage, and is surrounded by a more or less distinct cyst wall. The nuclear structure here

also is characteristic. The cyst contains several (from two to eight, depending upon the species of entameba and the stage of development of the cyst) ring-form nuclei, usually smaller than, but of the same structure as, the nucleus of the motile entameba.

Walker summarizes the differential points between *Entameba histolytica* and *Entameba coli* in a general way as follows:

MOTILE STAGE

A. *Entameba histolytica.*	B. *Entameba coli.*
1. Appearance hyaline.	1. Appearance porcelaneous.
2. Refractiveness more feeble.	2. Refractiveness more pronounced.
3. Movements active in the fresh stools.	3. Movement sluggish.
4. Nucleus more or less indistinct.	4. Nucleus distinct.
5. Chromatin of nucleus scanty.	5. Chromatin of nucleus abundant.

ENCYSTED STAGE

A. *Entameba histolytica.*	B. *Entameba coli.*
1. Cyst smaller.	1. Cyst larger.
2. Cyst less refractive.	2. Cyst more refractive.
3. Cyst usually contains elongated refractive bodies known as "chromidial bodies."	3. Cysts do not contain "chromidial bodies."
4. Nuclei never more than 4.	4. Nuclei 8, occasionally more.
5. Cyst wall thinner.	5. Cyst wall thicker.

Willets states that the difference in size of the cysts of the two species is very helpful in making a differential diagnosis. Size alone, however, is not dependable, for the reason that small *Entameba coli* cysts are found at times. Large four-nucleated cysts have not been seen by me unless encystment was obviously incomplete. Since the nuclei lie in different planes, doubt occasionally exists as to the number of nuclei present in a given cyst. By using gentle pressure on the cover-glass the cyst wall can be broken and the escaped nuclei, lying just outside of the cyst wall, counted. Another practical point in differentiating entamebic cysts is the manner in which a preparation is made for examination. It is his custom to make a thin preparation and to draw off the excess water with a piece of filter-paper so that the cover-glass fits snugly to the

specimen. Thus the cysts are flattened out somewhat and the counting of the nuclei facilitated. This manner of making a coverslip preparation also lessens the probability of the cyst shifting to another field when the oil immersion lens, which must always be used to make a reliable differential diagnosis, is applied.

Willets emphasizes that diagnosis of entamebic infection should be made not only upon the presence of active, but also upon that of quiescent or encysted forms. The one safe stage of development for a differential diagnosis between *E. coli* and *E. histolytica* is the encysted form encountered in formed stools.

(2) *Macroscopic Examination of Stool.*—The stools are commonly of a greenish-brown or yellow color and of a purée-like consistency, intermixed with flakes of mucus and food bits. They may be blood-streaked or foamy. The *reaction* is usually definitely alkaline. The odor is often quite characteristic—a peculiar penetrating, pungent mustiness that sometimes is definitely ammoniacal. Where there is much blood or necrotic tissue the foul, acrid, putrid odor is disgustingly nauseating.

(3) *Microscopic Study.*—The most striking feature of the stools, apart from the parasites, lies in the presence of enormous numbers of large and small motile and non-motile bacilli or spirilla. Indeed, such great numbers of bacteria often urge one on to examination of many stool specimens for protozoa when the first slides have proved negative. Frequently the bacteria are so numerous as to resemble a hanging-drop from a bacterial culture.

In addition to bacteria are generally noted actively budding yeasts, chains, or groups of cocci or of torulæ, scattered collections of mucous corpuscles or leukocytes, and occasional partly degenerated red blood-cells. In some smears ammonium triple phosphate crystals and leucin plates and fatty acid needles are often very abundant. There are generally considerable undigested vegetable tissue and partly broken up muscle bundles when the patient has been on fairly full diet.

(4) *The Protozoa.*—As already stated, persistent search is often necessary before the organism can be properly seen and

identified. This applies particularly to entamebæ; the flagellates are readily recognized by one with the common training in parasitology. There is still considerable debate respecting the identity of Entameba histolytica and tetragena. We have followed the classifications of Darling and of Craig in considering the cellular variations to represent different stages of the same organism. The histolytica stage appears to represent the organism at its maximum of activity and probably pathogenicity.

In our series of cases the incidence of the protoza was as follows:

TABLE IV

Cercomonas intestinalis hominis	36
Trichomonas intestinalis hominis	21
Entamebæ—	
Histolytica stage	17
Tetragena stage	6
Unclassified	4
Amebæ total	27
Lamblia intestinalis hominis	5
Megastoma entericum	2
Balantidium coli	2
Grand total of cases	93

COINCIDENT INFECTIONS

Cercomonas with Entameba	12
Trichomonas with Entameba	14
Cercomonas, Trichomonas, and Entameba	4
Trichomonas and Cercomonas	8
Cercomonas and Lamblia	3
Cercomonas, Trichomonas, and Lamblia	2
Trichomonas, Cercomonas, and Megastoma	1
Trichomonas and Balantidium	2

(5) *Chemical Examination of the Stools.*

(*a*) *Blood.*—Altered blood was demonstrated in 74 instances (79 per cent.) by the benzidin test.

(*b*) *Pancreatic Efficiency Tests.*—Quantitative estimation of trypsin and amylase was made by the methods of Gross-Fuld-

Wohlgemuth in 31 cases. In 22 instances (70 per cent.) trypsin was reduced below the normal (500 units). In 9 instances (29 per cent.) amylase was deficient. (Figs. 182, 183, 184.)

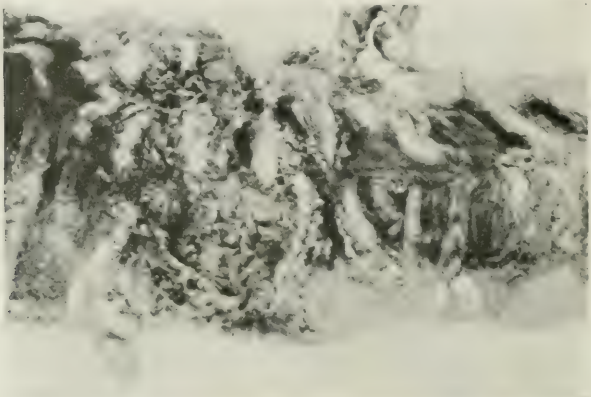


Fig. 182.—Specimen from colon, showing acute, ulcerative amebiasis. (After McDill.)

(c) *Bile-pigment.*—By the Schmidt bichlorid test made upon stools from 46 individuals bilirubin was present in 32 (69 per cent.) and biliverdin in 14 (30 per cent.).

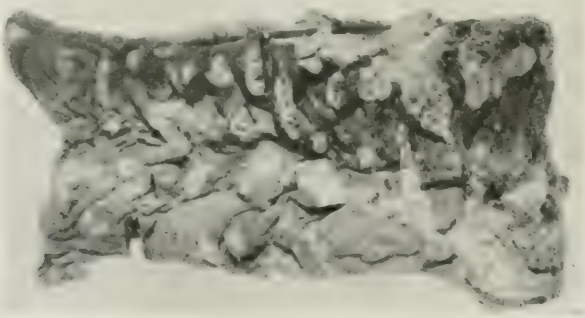


Fig. 183.—Specimen of colon, showing multiple areas of necrosis in acute amebiasis. (After McDill.)

(6) *Gastric Analyses.*—It is not infrequently stated that where protozoa are found chronically in the stools of patients of the temperate zone such are secondary contaminations due to gastric achylia. It has not been shown why, if such be the

case, there should not be a more universal finding of protozoa in the great group of achylias that come under observation. It is not unlikely that the rapid multiplication of protozoa and symbiotic bacteria in the intestinal canal is capable of bringing about toxic gastric or pancreatic achylias. Similar occurrences have been noted in association with typhoid fever and pellagra in our clinic and in cholera and intestinal tuberculosis by others.

In our series there were 40 cases (43 per cent.) with gastric achylia, 33 cases (36 per cent.) with subnormal hydrochloric

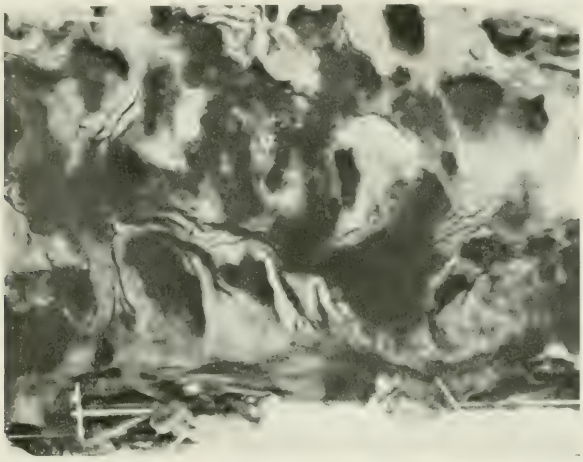


Fig. 184.—Colon in Balantidium coli infection. (After Bowman.)

acidity, and 20 cases (21 per cent.) with normal or increased gastric HCl. In one instance of most pronounced acute infection with cercomonads and trichomonads the free HCl was 86.

The gastric motility was normal in 83 cases (89 per cent.). In 10 cases (10 per cent.) there was mild stagnation.

(7) *Anemia.*—In 19 instances this was sufficiently striking to suggest pernicious anemia. Study of numerous blood counts and films failed, however, to establish the disease. The characteristic anemia is of secondary type, such as is common in chronic intoxications – viz., nephritis, cancer, or malnutrition. The average *hemoglobin* was rather more than 70 per cent.

The average red cell count was 3,120,000. The average white cell count was 8400. The differential counts revealed no great abnormality apart from the tendency of the small lymphocytes to increase at the expense of the polynuclears, and, in some instances, a rather high percentage of eosinophils. There were 69 cases (74 per cent.) where the eosinophil percentage equalled or exceeded 3. There were 11 instances where it ranged between 8 and 14 per cent. In 1 case the eosinophils reached 18.5 per cent. (an instance of amebiasis and trichomoniasis). The blood counts are apt to show higher eosinophil counts before purgation than afterward. It was not observed that blood from cases presenting clinical signs of grave intoxication have the highest eosinophil counts; in fact, the most pronounced eosinophilias were recorded in young individuals who had presented symptoms of protozoic infection for but a few years.

(8) *Urine.*—In 16 instances albuminuria was noted. It is not possible to state that this finding was directly due to the presence of parasites. Many of the cases were at the age in life when traces of albumin are quite commonly found in the urine.

Treatment.—(1) *General.*—It is important that all local infection foci (teeth, tonsils, mouth or throat ulcers, diseased gall-bladders or appendices, etc.) should be removed before attack is made upon the intestinal infection. If such are not taken care of radically, reinfections may occur or subsequent ailments of such parts may lower general resistance sufficiently to again permit of enteric infection by protozoa. Encysted protozoa may lurk for years in the appendix or the gall-bladder. When such is the case the host is to be considered a not altogether harmless carrier.

(2) *Measures to Free the Intestine of Protozoa.*—A preliminary preparation of the intestinal canal enables one to most quickly bring about its relative sterilization. In my clinic these patients are placed upon liquid diet for two days before medical care is begun. This permits of the bowel being freed from firm residues. Each morning they receive a glass of citrate of magnesia solution. *The aim of specific medicines* is (*a*) to render inert protozoa infecting the intestinal contents, and thus prevent infection of the mucosa, and (*b*) to destroy organisms already lodged within

the mucous membrane. Successful therapy depends upon proper isolation of the infecting parasites—entamebæ are particularly susceptible to ipecac or its alkaloid, emetin, while flagellate or ciliated protozoa are slightly affected by these drugs, but are readily destroyed by calomel. Thymol is effective against both parasites.

(*a*) In the *entameba cases* the patient is put to bed on liquid diet, with hot pads moistened in boracic-alcohol mixture over the abdomen (to prevent colicky pains or abdominal discomfort). He is then given by mouth a 10-grain tablet of the aluminum salicylate of ipecac ("alcresta") every hour and $\frac{1}{3}$ grain of emetin hydrochlorid hypodermically every four hours for two days. If the stools show diminution of the parasites the dose of ipecac and emetin is then reduced by one-third, and this continued for another two-day period. No reduction is made if the parasites are still very abundant or are very active. Usually by the end of the first week the patient is taking emetin, $\frac{1}{3}$ grain, hypodermically, twice daily, and 10 grains of ipecac ("alcresta") four times daily. The treatment is continued even when no parasites are seen. Accompanying the medicines given by mouth, the colon is carefully lavaged with 4 quarts of hot normal salt solution or a solution of quinin (1 : 3000) and thymol (1 : 5000) in normal salt solution night and morning. On the sixth day the patient is put on fat-free diet for twenty-four hours. At bedtime of the seventh day 30 grains of thymol in honey are administered at 8 P. M. and again at 10 P. M. At 6 A. M. the following morning the patient gets 2 ounces of Epsom salts in hot water, and all that morning frequent drinks of black coffee, fat-free broth, or malted milk. During the second week, the emetin, ipecac, and bowel irrigations are continued, and usually on the tenth day from the beginning of the treatment two doses of 15 grains each of thymol (preceded by twenty-four hours of fat-free diet) are given in the evening. Daily examinations of the warm stools usually indicate no parasites by this time, and the diet may be increased according to the patient's desires, provided it is low in protein and not very bulky. If parasites persist at the end of two weeks, then, after thorough colon lavage with hot normal saline solution, from 500 to 1000 c.c. of filtered

commercial kerosene are given per rectum, slowly. The external parts are greased with carbolated vaselin and effort is made to have the patient retain the kerosene for at least one hour. We have never seen any harmful effects follow the use of kerosene. It has proved very efficacious in ridding the bowel of persistent infection (entamebæ and lamblia).

When the entamebæ are no longer demonstrable in the freshly passed stool, then local treatment of the enterocolitis by large doses (30 grains) of bismuth subnitrate or salicylate given five times daily should be carried out. Emetin and ipecac should be continued for at least six weeks; the ipecac alone for three months. The bowel irrigations are usually stopped at the end of the third week. The general state of the patient is taken care of according to indication—HCl after meals if the gastric juice is lacking in acid; iron and arsenic if anemia is present.

(*b*) When *flagellate protozoa* are the infecting organisms the treatment is substantially as outlined above for entamebæ except that emetin and ipecac are not used unless there is a concomitant amebiasis. The flagellates are readily destroyed by the administration of evening doses (5 to 15 grains) of calomel followed by 2 ounces of Epsom salts the next day. These doses of calomel are repeated about every five days, according to the indications furnished by the stool examinations. The flagellates are usually less persistent than the entamebæ (with the possible exception of *lamblia*).

(*c*) *After-treatment.*—Our study of specimens of gall-bladders and appendices removed at laparotomy indicates that in these parts of the gut cysts of protozoa may lurk for years. Reinfection of the bowel is thus possible. Consequently, if these organs have not been removed, we insist that our patients have stool examinations at least three times a year, and that they go through an abbreviated course of treatment similar to that above outlined. Only in this way do we believe that protozoa "carriers" can be eliminated or reinfection of so-called "cured" cases prevented.

NOTE.—I wish to express my thanks to Miss F. Colson and Doctors Bennett Parker and M. McRae for photographs and sketches.

CLINIC OF DR. M. MILTON PORTIS

Cook County Hospital

CARCINOMA OF THE ESOPHAGUS

Discussion of the Pathology and Clinical Picture; Röntgen Ray Preferable to Esophagoscope in Establishing the Diagnosis; Possible Dangers in the Use of Sounds or Bougies in Esophageal Stricture; Treatment Chiefly Palliative.

CARCINOMA OF THE ESOPHAGUS

Outline

- Occurrence and Predisposing Causes.
- Pathology
  - 1. Varieties
    - Primary.
    - Secondary.
  - 2. Size.
  - 3. Location.
  - 4. Metastases.
- Symptoms
  - Early.
  - Late.
- Diagnosis
  - Clinical picture.
  - Sounds.
  - Esophagoscope.
  - *x*-Ray.
- Differential Diagnosis.
- Complications
  - Local extension.
  - Perforation.
  - Metastasis.
- Prognosis and Cause.
- Treatment
  - Medicinal.
  - Mechanical.
  - Diet.
  - Surgical
    - Removal of growth.
    - Gastrostomy.
  - Radiotherapy.

Case I.—Patient is a widower, age seventy years; occupation, laborer; nationality, Irish.

Present Complaint.—Regurgitation of food; pain over epigastrium, burning in character; loss of weight; weakness and faintness.

Previous History.—He has never been sick until the onset of the present illness except for an occasional cold and asthma, which began nine years ago and continues at the present time. Nine years ago he also had necrosis of the jaw from defective teeth.

Present History.—The onset dates back to four months ago, when he began to complain of inability to retain food and water, which was promptly regurgitated. He is at times able to retain an eggnog and some milk, but all other food comes back quickly. His mouth fills up, and on opening it the contents are expelled. At the time of the onset he had a severe hemorrhage. The blood rushed into his mouth, was bright red for the most part, and contained some clots. He had another hemorrhage of the same type last night, when the blood rushed from his mouth in a stream and with passage of considerable clots.

He has had a cough for years, which now is somewhat worse and quite loose. There is some sputum. The material he raises by coughing is usually white and frothy, but sometimes brown and bitter. He cannot lie on his back or right side because his coughing becomes worse and he has smothering attacks. He has lost 37 pounds in weight since the onset of the present trouble. He has been very weak and actually fainted in the examining room today.

Habits.—Drank considerable beer and whisky; heavy coffee drinker. Bowels have been very inactive.

Venereal diseases denied.

Family History.—Father died of heart trouble; mother, of gastritis. Two sisters dead, cause not known.

Physical Examination.—Inspection shows an emaciated, weak, adult male, about seventy years of age, pale and somewhat yellowish in color.

Head and Scalp.—Scar on back of head over occipital region from injury; ears and nose negative.

Eyes.—Pupils small, react to light and accommodation. Conjunctivæ very pale.

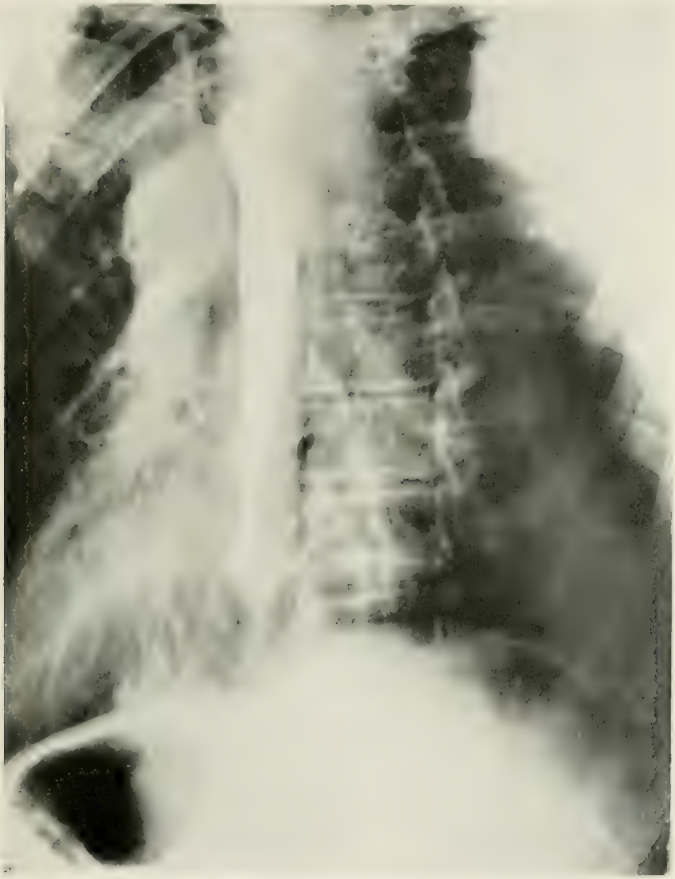


Fig. 185.—Carcinoma of lower esophagus involving lower one-third down to cardiac end of stomach.

Mouth.—Teeth remaining are very bad. Tongue coated. Pharynx pale.

Neck.—No tumor masses or adenopathy.

Heart and Circulation.—Radial pulse fairly good, but some-

what irregular. No thrills. Apex-beat of good quality, circumscribed, and in nipple line.

Heart Borders.—Left border 2 cm. outside of nipple line. Right border in right sternum.

Tones.—Very weak, but distant.

Respiratory System.—Chest symmetric; respiration normal; fremitus equal over both lungs. Hyperresonance on percussion. Lung borders limited in excursion. Many râles of all kinds; loud, dry, and piping râles present over anterior part of chest, especially on right. Breath sounds less prominent posteriorly; moist râles present.

Abdomen.—Sunken and doughy. Pulsations of abdominal aorta visible; liver palpable 3 inches below costal margin. No tumor masses. Spleen not palpable.

Skin.—Very white, dry, lifeless, and inelastic, with no adipose tissue.

Extremities.—Thin and emaciated.

Reflexes all normal.

x-Ray Examination by Dr. Blaine.—Carcinoma of upper part of esophagus directly behind the aorta.

Esophagoscopic Examination.—Patient has obstruction about 11 inches from teeth, where an irregular, pale growth could be seen.

Urine Examination.—Negative.

Blood.—Negative.

Wassermann Test.—Negative.

Case II.—Patient is a laborer; nationality, German; age sixty-four years.

Present Complaint.—Expectoration of blood; pain in chest and upper abdomen; loss of weight.

History of Present Illness.—Patient states that his illness began two months ago. He makes rather indefinite statements concerning his illness, but states that at first he had pneumonia. The symptoms which he describes are difficulty in breathing and pain in the chest. The history of coughing is indefinite. He also has pains in the epigastric and right hypochondriac regions. He was treated by a physician and his pain in the chest was

relieved for a time. His appetite has been poor, and in the past four or five months he has lost over 40 pounds in weight. In the past six or seven weeks his condition has become worse and he has vomited after eating. One week ago he began to expectorate blood. He states that at the first hemorrhage he expectorated five basinsful of blood. The next day he expectorated two basinsful. Since he has been in the hospital he has expectorated about 50 c.c. of bright red blood and fresh clots. Mixed with the blood is a considerable amount of tenacious mucus.

Past History.—No illness before; always in good health; denies venereal disease.

Habits.—Drinks a pint of beer daily. No whisky.

Marital History.—Wife and seven children living and well.

Physical Examination.—Patient is an adult male, sixty-four years of age, who complains of pain in the chest and upper abdomen. He expectorates small quantities of fresh red blood mixed with mucus; the blood apparently comes from the throat and upper air passages.

Skin.—Negative, except for its laxity and dryness.

Head and Neck.—No deformities of cranium. Scalp negative; ears and nose negative.

Eyes.—React to light and accommodation.

Mouth.—Tongue shows a slight grayish coat. Teeth discolored and carious. Tonsils small, no injection or exudate.

Pharynx.—On examination of the pharynx blood can be seen bubbling up from the throat.

Chest.—Symmetric; expansion equal. On palpation he complains of indefinite tenderness over the chest posteriorly and extending downward anteriorly over the upper abdomen on the right side.

Lungs.—There is impairment of resonance over the right upper lobe posteriorly with bronchovesicular breathing. Left lung negative.

Heart.—Apex-beat three fingers wide, located in fifth interspace in the nipple line. The beat is forcible and heaving and a thrill is palpable.

Boundaries.—Right, 4 cm. from midline; left, 12 cm. from midline.

Sounds.—The first tone at the apex is loud and snappy and is preceded by a short presystolic murmur; a soft systolic murmur is audible at the pulmonic area; aortic and tricuspid areas negative. Pulse 88, somewhat irregular in rhythm and volume; moderate tension.

Blood-pressure.—Systolic, 108; diastolic, 55; pulse pressure, 53.

Abdomen.—Normal in contour. No distention or rigidity. There is tenderness over the right upper quadrant on deep pressure. The liver extends 5 cm. below the costal margin and is tender to the touch.

Spleen and kidneys are palpable. No fluid or tumors.

Genitalia negative.

Extremities.—Tissue soft and flabby and the skin relaxed, indicating loss of flesh. Skin is dry. No edema or adenopathy.

Blood Examination.—Red blood count, 3,380,000; white count, 16,000; Hgb., 62.

Wassermann Test.—Negative.

Urine Examination.—Straw colored; reaction, acid; no abnormal findings.

The *x-ray examination* shows the picture of a carcinoma in the upper third of the esophagus.

Etiology of Carcinoma of the Esophagus.—A carcinoma is the most common disease of the esophagus. Keep in mind that of carcinomata in all parts of the body, one-fifth will be found in the esophagus; 80 per cent. occurs in males usually beyond middle age. Whereas other carcinomata are apt to occur around the forties, esophageal cancer is apt to develop in the sixties and later. Curiously enough there is often no preceding history of stomach trouble at all. Usually there is a history of alcoholism or of excessive use of tobacco, but just what rôle these play we do not know. Sometimes the patient gives a history of trauma of the esophagus by sharp, hard food. In other cases the patient tells of a habit of taking his food and drink quite hot and thus irritating the esophagus.

Pathology.—*Varieties.*—The common variety is epithelioma and the tumor is usually a primary one. Secondary tumors of

Fig. 186.—Carcinoma of esophagus at level of bifurcation of trachea. Dilatation above stricture (Blaine).

the esophagus do occur rarely, but they are more apt to be tumors pressing on the esophagus.

Size.—The size of the primary tumor is variable, depending on its age and extension. Sometimes the mass is very small and involves one side only; at other times it is as large as an apple and surrounds the esophagus, while in still others it may involve the entire length of the esophagus. Colloid carcinoma occurs, but is rare, just as it is rare in other parts of the body.

Location.—Usually the growth has a single location. When there is more than one tumor in the esophagus it is either the rare occurrence of two growths at the same time or, more commonly, it is an implantation of the first growth in some other place in the esophagus. This indirect inoculation can be made by a sound causing trauma and implanting some carcinoma cells in other parts of the esophagus. Sometimes in the act of swallowing a carcinomatous fragment going over a raw surface may implant itself. The carcinoma frequently encircles the entire esophagus and then grows on to the surrounding tissues, but at times it does not quite encircle the entire esophagus. If one-fifth of the circumference of the esophagus is uninvolved, such an individual may have normal swallowing by compensatory dilatation of this one-fifth of the esophagus. Once the growth surrounds the esophagus then symptoms of obstruction naturally start.

Sites of Predilection.—The sites of predilection are those where there is a physiologic narrowing. Such locations normally offer resistance to the swallowing of food. These are found opposite the cricoid cartilage, the bifurcation of the trachea, at the diaphragmatic opening for the esophagus, and at the cardia. If irritation plays any rôle, it would be at such places that friction would be greatest. One group of statistics gives the commonest location at the bifurcation of the trachea, and another group limits it to the upper third of the esophagus. The majority will be found in the upper half, which would include the region of the bifurcation of the trachea. The esophagus usually dilates and sometimes hypertrophies above the obstruction. The lower the stricture, the greater the dilatation because food remains longer.

Metastases from carcinoma of the esophagus are uncommon, but they do occur. Glandular involvement is very common. In many cases rather early the left supraclavicular lymph-glands can be palpated. The tumor invades the surrounding tissue, and by direct growth and extension frequently interferes with the function of the organs of the chest. Mediastinal involvement is usually the greatest. The tumor mass may press upon the aorta; it may press upon the lung and invade the pleura; it may even press upon the heart. Fistulous tracts in the carcinoma may form into any of these organs or places and thus rupture into the pleura, lung, or trachea, into a bronchus, into the aorta, or into the pericardium. Pressure of the growth upon the spine also occurs, causing erosion of the spine and involvement of the spinal cord, with serious if not fatal manifestations.

Symptomatology.—When you understand the pathology of the disease the symptoms are rather easy to determine and to explain. Most of these cases occur in old men who have previously had no stomach trouble, and the first thing that they will complain of will be difficulty in swallowing. That is the complaint that will bring them to the doctor. This difficulty in swallowing is at first manifested in the taking of coarse foods or eating the food rapidly, so that large pieces are swallowed. They will tell you that they are accustomed to take large drafts of liquid to wash it down. Later, as the obstruction increases, even liquids cause difficulty in swallowing. Sometimes the patient thinks the trouble is in the pharynx, and at other times will complain of trouble in the stomach. This depends on the location of the carcinoma. In the early stage they usually have no pain or regurgitation, but when the obstruction increases then regurgitation begins—at first of the solid foods and later even the liquids. The patient this morning vomited liquid that he had taken an hour before. The amount that is regurgitated and the time of the regurgitation depends upon the location of the growth and upon the amount of dilatation above the growth which occurs secondarily. The lower the obstruction, the later the time of regurgitation, and likewise the more the

dilatation, the later the time of regurgitation. The amount regurgitated will vary with the dilatation and the amount retained.

The food brought up in the earlier cases is practically unchanged, but later, when the tumor has ulcerated, foul-smelling material may come up containing tumor fragments and purulent or bloody material. Then, too, usually associated with this there is a good deal of pharyngitis, and a thick ropy mucus comes up which causes a great deal of difficulty in bringing up the regurgitated material.

Later in all the cases pain comes on and is a variable factor, depending upon the amount of ulceration and also upon the location of the growth. The higher the growth, the greater the pain.

If you watch these cases under the fluoroscopic screen you can see at times violent peristaltic movements of the esophagus in the effort to push food through the obstruction. With these violent efforts the patient complains of pain. The ulceration of the growth, particularly if the ulceration is near the mediastinum, will cause pain, which may be of a constant boring type, but usually the pain is intermittent and is associated with the retention of food. Once the food is regurgitated the pain disappears, to occur again on the ingestion of food.

If the patient tells you that he has intermittent trouble in swallowing you have to keep in mind one of two things—either large pieces of food, causing temporary obstruction in the narrow opening of the esophagus, so that a plugging occurs from time to time, or ulceration is occurring and the tumor channel is enlarged for a time only and will narrow down again.

In all of these cases cachexia, marked emaciation, and loss of strength occur, due not only to inanition, which is a marked factor in these cases, but also due to the toxic absorption from the carcinoma itself. You have a double reason for the rapid loss of weight and strength in these patients.

Complications.—When the tumor extends to surrounding organs or metastasis occurs the patient may complain of a great many things. There may be mediastinal disturbance, pressure

on the lungs with dyspnea, pressure on the recurrent laryngeal nerve, causing hoarseness by paralysis of one of the vocal cords and a peculiar brassy cough. The tumor may press on the sympathetic and cause an inequality of the pupils. If the spine is pressed on and eroded corresponding nervous symptoms and findings will occur.

Diagnosis.—In the diagnosis a history of obstruction and difficulty in swallowing in an old man, without a history of syphilis and without a history of trauma to the esophagus, is most significant. Practically 90 per cent. of such cases are carcinoma of the esophagus.

Differential Diagnosis.—Carcinoma of the esophagus must be differentiated from all other conditions producing obstruction in the esophagus. Benign strictures of various kinds, particularly those due to the swallowing of caustics, syphilitic stenosis, aneurysm pressing on the esophagus, mediastinal tumors of various kinds pressing on the esophagus, ulcer of the esophagus with stricture, hysteric conditions producing spasm of the esophagus, abscesses originating in the spine, tubercular abscesses pressing on the esophagus, kyphosis of the spine usually due to tuberculosis, and a host of other things all come up for differential diagnosis. But when you weigh your case and have studied it from various phases, particularly from the röntgenologic side, the diagnosis becomes comparatively easy. If you are convinced that there is no aneurysm present, you may pass a stomach-tube. If it meets with an obstruction, then the question comes up whether or not you should put down a bougie or whether you should use the *x*-ray immediately. By all means use the *x*-ray if it is accessible. I consider the use of a bougie or sound for the diagnosis of stricture of the esophagus to be exceedingly dangerous. I use them only in treatment, and then only on a swallowed string as a guide.

Röntgen Examination of the Esophagus.—The esophagus lends itself for *x*-ray examination particularly well. Have the patient swallow food or liquid containing some barium or bismuth, and on the fluoroscopic screen a very definite image of the esophagus and the place of obstruction can be seen. In making

such observations it is best to place the patient standing in an oblique position, so that the spine and the heart can be distinguished from the esophagus. In this position you can see beautifully and distinctly the opaque mass traveling down the esophagus. The place of obstruction, the amount of dilatation above, the length of the obstruction, the surrounding involvement, are all very apparent, and a picture can be taken just as the patient is swallowing. The screen is, however, very much

Fig. 187.—Carcinoma of upper esophagus with dilatation above stricture.

better than an individual plate. When you recognize the irregular outline just above the stricture and the zigzag course of the stricture proper, you can readily understand how a bougie could make a false passage. It is a very easy thing to do. The tumor is soft, and the bougie will get in some one of these irregular places, and without very much pressure will produce perforation.

Indications for Use of Esophagoscope.—The esophagoscope, which was used a great deal until the *x*-ray came into vogue,

was a splendid way of determining the nature of the disease, but esophagoscopic work is rather hard upon the patient without an anesthetic. With this instrument you can get a view of the tumor just at the place of narrowing. You cannot estimate, as with the *x*-ray, the extent of the tumor. Nor can you determine whether there is anything outside of the esophagus. The *x*-ray will show if the patient has, in addition to the carcinoma, an aneurysm without having done the patient any harm. In doubtful cases the esophagoscope can be used for inspection and to procure a piece of the tumor for diagnosis. *Let me urge you again in esophageal cases by all means to use the x-ray in making a diagnosis if it is at all accessible;* if you have not a fluoroscope, take some plates.

The **prognosis** is absolutely bad in all cases. No case of cure has come to my notice. Usually the course is rapid, lasting only months, but it may last one or two years. When perforation into any of the surrounding organs occurs death usually follows. The left bronchus and the aorta are the common places where perforation occurs.

Perforation into the lung is usually followed by gangrene.

Treatment.—(1) *Palliative.*—The treatment should be considered from the medicinal side, the dietetic side, the mechanical side, and the surgical side. The use of drugs in carcinoma of the esophagus as a curative measure of course does not accomplish anything, but it does alleviate the suffering of the patient and at times prolongs his life. Opiates should be used for the pain in this disease. Frequently local applications of cocain or the swallowing of a cocain mixture will be of benefit in the control of pain. At times a cocain mixture can be injected into the tumor itself by the use of a perforated bougie. We do not resort to such methods very often, but we do give opiates by mouth or hypodermically for the control of the pain. Inasmuch as the diagnosis of carcinoma of the esophagus always leaves a little doubt concerning syphilis, even with the negative Wassermann, I think it is wise to give the patient a course of mercury and of iodids. It cannot do any harm and it may do good.

(2) *Dietetic.*—Patients should be fed with semifluid or fluid articles of diet in the early stages before obstruction is marked. Choose nutritious foods, as milk, eggs, eggnog, soft-boiled eggs, the various soups, and the purées of various vegetables. Omit from the diet the solid foods that would cause difficulty in swallowing.

Fig. 188.—Carcinoma of esophagus involving upper two-thirds. Ulceration into aorta. Below, gastrostomy opening with skin section attached (Le Count).

(3) *Mechanical.*—When the patient can no longer swallow liquids, then it is a question what is the proper course to pursue to maintain the nutrition of the patient. In some cases, with the intelligent co-operation of the patient, it is wise to advise a gastrostomy. Through the gastrostomy opening the patient can be fed with anything and everything until the tumor itself causes the death of the patient. Many patients, however, refuse to have gastrostomy done, and in some it is not wise to tell the patient the nature of the trouble.

Without gastrostomy, what are you going to do? There are bougie catheters or perforated bougies that can be inserted into the stricture of the esophagus, and this is best done on a string, as Plummer first pointed out. The patient swallows

several feet of heavy silk, which enters the stricture. Upon this heavy silk you can thread your bougie, and by keeping the string a little taut you can guide the tip of the bougie to the

Fig. 189.—Same specimen as Fig. 188, showing perforation into aorta at *A* (Le Count).

opening, and in that way run much less risk of perforation. Do not use any force. If sounds are passed for dilating the stricture, use the same string method, but do not dilate oftener

than every other day. When such a perforated bougie is in place, you can feed the patient with various foods that we have spoken of. Sometimes we leave these bougies in place for several days, and at other times they are introduced only at times of feeding. You assume the risk of perforation, but when you consider the nature of the disease what is the harm of such danger? You might cut life a little shorter by accident, but if you can give your patient comfort and feed him it is a wise thing to do. I have fed patients in this way for months. When you can no longer get through with any type of instrument, and you do not want to do a gastrostomy, then you have to resort to rectal feeding. When this begins, life is limited, but do not let your patient suffer for lack of water. Use hypodermoclysis if necessary. Extreme thirst and suffering from want of water is a punishment that a patient should not be subjected to.

(4) *Surgical.*—In surgical treatment the aim should be the removal of the growth. This has been attempted through the chest, but without good result. With the proper type of respiration apparatus or respiration chamber for operating upon the chest such operations are carried out, but are rarely successful.

Then comes the question of gastrostomy, which is easily accomplished even in a weakened patient under local anesthesia. Through the gastrostomy opening the patient can be fed with all sorts of food and any drug can be given.

(5) *Radiotherapy.*—In selected cases deep Röntgen therapy should be given a trial, and the possibility of applying radium directly to the tumor by means of a bougie should also be considered.

I shall now show you a specimen from the Rush Medical Museum showing carcinoma of the esophagus involving the middle and upper thirds which is just about to perforate into the aorta (Figs. 188, 189).

CLINIC OF DR. CHARLES SPENCER WILLIAMSON

COOK COUNTY HOSPITAL

A CASE OF HEMOPNEUMOTHORAX

Mode of Onset. Repeated Aspiration of Hemorrhagic Fluid from the Chest. Tuberculosis the Probable Etiologic Factor.

February 28, 1917

THE patient whom I wish to show you today is one who has been under my observation for a long time, and the condition in which he finds himself today is the direct outcome of his original disease, although he is now in a very different condition from that in which I first saw him.

He is, as you see, a young man rather remarkable for his height, something over 6 feet 2 inches, and his slimness, his weight now being about 145 pounds. He is of German-American parentage, born in this country, and a broker by occupation.

The *family history* is substantially negative, in that his mother and father are both living and well and show no evidence of any hereditary disease. He has brothers and sisters also living and well. There is no history of tuberculosis in the family.

His *past history* is fairly uneventful. He had the ordinary diseases of childhood and an attack of appendicitis at twenty years of age. He has always considered himself of a highly nervous temperament, and I can fully corroborate this. I have rarely ever seen a patient who more thoroughly deserves the term "neurotic." I mention this particularly because in the evaluation of his symptoms it is rather important to bear in mind the strong tendency he has to exaggerate them and the tendency, equally great, to manufacture symptoms. He would

really make a fairly interesting neurologic clinic, and I fancy that he might be used as an instance of half a dozen "phobias."

Marital and Venereal History.—The patient is unmarried and denies venereal infection of all sorts. The Wassermann reaction was negative, as was also the complement fixation for gonorrhea.

Present History.—Up to the onset of his present illness the patient insists that he was perfectly well except for a number of symptoms largely having to do with fear of various things, and which he attributed, I think very correctly, to his own neurotic state. He had no cough, and while he had always been, as he expresses it, of a "bean pole figure," he had not been losing weight and had had an excellent appetite. His occupation as a broker on the Board of Trade carried with it a great deal of mental excitement and worry. He lives at an uptown hotel, and his life was going along in its customary manner until late one afternoon, after coming home from his office, he stopped in the lobby of the hotel for a moment, and, being thirsty, bent over to take a drink of water from a faucet, and as he did so felt, as he said, "something crack" in the left side of his chest. It caused him a little pain, but not a great deal. He stood around the lobby for a few minutes, and then got into the elevator to go to his room. While in the elevator he was attacked with vertigo, things grew black before his eyes, and he told the elevator man that he was fainting. He was helped to his room and laid across the bed, feeling very much nauseated, weak, and dizzy. A neighboring physician was sent for, came over and made an examination, but could not come to any definite conclusion. The temperature was taken and found to be subnormal. The pulse was very rapid, about 140 to 150, weak, and thready. The physician, who lived just across the street, arrived within a very few minutes of the occurrence, and I feel sure that his statement can be relied upon as representing his condition at that time. Some stimulants were ordered and a brisk cathartic, but the patient seemed in a very critical condition all during the night, and the following morning his condition was so alarming that the physician who was first called requested a consul-

tation. This consultation took place the following morning, and both physicians agreed that there was something acutely wrong with the left chest. I was called in with them an hour or two later, and on my arrival found the situation as follows: The patient, still with most of his clothes on, was lying on his left side diagonally across the bed looking as if death were imminent. He was pale, the extremities were cold and clammy, the respirations very rapid, approximating 42 or 44, the pulse barely perceptible and so rapid as to be counted with difficulty. The temperature was 96.8° F., and the picture was one of the most profound shock—a duplicate of the picture one sees in profuse hemorrhages in typhoid and similar conditions.

Physical Findings.—The difficulties of the examination were quite considerable. The patient was far too weak to sit up or, indeed, to be subjected to a very critical examination. I proceeded to make such physical examination as was possible, and found that the eyes, ears, nose, and throat were negative and there was nothing abnormal about the face except the pallor and the cold, clammy sweat. There were no venous pulsations perceptible in the neck and the carotid pulse was feeble and exceedingly rapid. On examination of the chest, with the patient lying on his back, the first thing that was noticeable was that the left side of the chest showed a greatly diminished—indeed, practically absent—respiratory excursion. The patient's lean build made this strikingly apparent at a glance. No apex-beat could be seen or felt, but there was a distinct diffuse pulsation underneath the sternum and over to the right of it. Respiration on the right side was deep and distinctly labored. With the patient still on his back percussion showed a resonant note all over the right side and a somewhat hyperresonant note all over the left side. In the left axilla, toward the posterior portion and low down, a dulness could be found amounting to flatness. On turning the patient over on the side similar conditions were found in the back. The right side was breathing deeply, the lung moving freely, as determined by the change of note on deep breathing. On the left side there was practically no respiratory excursion, and the

note was somewhat tympanitic over that portion of the chest which was uppermost and flat in the dependent portion. The patient was then, with the help of my colleagues, placed in a sitting position, and the change was very striking. There was flatness behind and in the axilla for a distance of perhaps 4 inches from the base and above this a tympanitic note. Auscultation showed on the right side slightly harshened breathing; on the left side almost nothing could be heard. Breath sounds were so distant and faint as to be barely audible. Over the area of flatness no breath sounds at all could be heard.

Diagnosis.—Now I hardly need say to you that a picture such as this, with practically complete absence of the respiratory excursion on the one side, a tympanitic note, almost complete disappearance of the breath sounds, and with an area of flatness which changed very rapidly and very perfectly with the position of the patient, is practically pathognomonic of pneumothorax with fluid in the chest. A little gentle shaking gave a very well-defined succussion sound and the *signe de sou* could be elicited very beautifully. With the patient again on his back it could be demonstrated very clearly how the mediastinum was pushed to the right or, more properly speaking, how it was drawn to the right with the collapse of the left lung. The right edge of the absolute flatness was fully five finger-breadths to the right of the median line, with a well-marked pulsation just to the right of the sternum, presumably due to the right ventricle.

The diagnosis of pneumothorax was very promptly made, and it was equally clear that there was a considerable amount of fluid in the chest. The patient's condition was so critical that it was decided not to move him at once to a hospital, but to await developments. Within twenty-four hours he rallied very distinctly, and his condition became sufficiently improved so that at the request of my colleagues I had him removed to a hospital and took over the management of his case.

On arriving at the hospital his temperature was 97.8° F., pulse 124, respirations 26—a very great improvement over his condition of the day before. The thing which had struck me

from the very beginning was the great rapidity of the pulse, out of proportion to the frequency of the respiration, and reminding me of an internal hemorrhage. As soon as he arrived at the hospital I therefore made an exploratory puncture, and was not so very greatly surprised to find that the fluid obtained was, as far as appearance was concerned, practically pure blood. Of course it is not to be thought that this was actually the case, as whenever there is a hemorrhage into the pleural cavity the conditions are such as to make it almost certain that there is a local reaction on the part of the pleura, so that an exudate forms which mixes with the blood and the resultant fluid obtained by aspiration is in reality a mixture of blood and pleural exudate in varying proportions. The specific gravity of the fluid first obtained was 1035, and to the naked eye could not be told from blood. It is to be noted that on standing this fluid did not coagulate, which is a condition rather commonly seen when blood has been shed into a serous cavity, such as the pleura. It was decided that a removal of a part at least of the fluid was indicated and we removed at the first puncture about 1 liter, and estimated that we had removed approximately one-third or one-fourth of the total amount. The patient was distinctly improved by the aspiration, which was carried out with great slowness, taking about an hour to remove the quantity stated. This was done deliberately, as it was and is my opinion that in all similar conditions a very gradual removal of the fluid is by far the safest plan.

The patient improved rather rapidly, and we find him on the fourth day of the disease with a pulse around 105 and a temperature of 100.4° F. From this time on his pulse-rate ranged from 85 to 100, with a slight elevation of temperature in the evening. The urinalysis made upon admission showed a little albumin and nothing else of any consequence. There was a leukocytosis of 28,800, with a differential count of 87 per cent. polymorphonuclears, 9 per cent. large mononuclears, 4 per cent. small mononuclears. There were 5,280,000 red blood-corpuscles and a hemoglobin of 75 per cent. This count was taken immediately upon arriving at the hospital. You will notice that the

red count has shown no evidence whatever of the hemorrhage into the pleura. The reason for this is readily enough understood. When a patient has a hemorrhage the first effect of the hemorrhage is to diminish the total quantity of the blood. It is only after some time has elapsed that the blood shows the effects of this hemorrhage in its corpuscular make-up. In other words, it is not until after the blood commences to become diluted by absorption of fluid from outside tissues in an endeavor to regain its original volume that the effects of the hemorrhage are shown in the lessened number of red corpuscles per cubic millimeter. This has been demonstrated by experimental work on animals. I have made some experiments on dogs, taking the hemoglobin spectrophotometrically immediately before venesection and then at various times subsequent to it. It sometimes requires many days before the minimum amount of hemoglobin is reached.

The further course of this patient's case was in one respect uneventful. The leukocytosis gradually subsided, although it had not reached the normal when he left the hospital seventy-two days later, the white count at that time being 14,800, with a hemoglobin of 80 per cent. The interesting feature in the subsequent course is seen in the results of repeated aspirations of the chest. In all, twelve aspirations were made, removing each time a quantity varying from 12 ounces to 1 liter. The first three or four taps showed practically the same fluid as at first, the specific gravity remaining about the same. By the thirty-third day the fluid was not being formed so rapidly, and on this date we removed all the fluid we could get and obtained only 27 ounces, with a specific gravity of 1022, still looking like almost pure blood. On the forty-second day we again removed 25 ounces, with a specific gravity of 1017, this being the last aspiration made. The fluid at this time showed a very marked change from the previous fluid in that it was no longer like pure blood, although, of course, still strongly hemorrhagic. Up to the last four or five specimens none of the fluids showed any clotting worth speaking of.

When we discuss the *diagnosis* of this case there are two

things which are perfectly evident. First, that he developed with great suddenness a pneumothorax, and, second, that he had a profuse hemorrhage into this pneumothorax, making the combination of hemopneumothorax. In the presence of such typical signs as I have already given you it will hardly be necessary to justify the diagnosis of pneumothorax by any further diagnostic considerations. The fact that he had a large hemorrhage is, of course, abundantly proved by the results of the aspiration. With the establishment of a hemopneumothorax, however, our diagnosis cannot be regarded as made. It is highly important to determine if possible the etiology, as the pneumothorax and the hemorrhage are, of course, merely incidents in the pathologic process, except in those extraordinarily rare instances in which a previously normal lung is supposed to rupture from overexertion. I may say right now that I, personally, very greatly distrust all the cases which have been reported of this variety. It seems to me practically impossible that a really normal lung should, with any exertion of which the patient is capable, actually rupture, and still less possible that it would rupture to such an extent as to tear a good-sized vessel and produce such a hemorrhage as we had in this case. I would feel unwilling to accept such an explanation unless actually demonstrated by an autopsy. My reason for making this statement is that many of the pathologic conditions may be so slight as to defy detection after the pneumothorax, and possibly even if one had had an opportunity of examining the patient beforehand.

Now let us ask ourselves what conditions might come into consideration. Beginning with the commonest, we find that tuberculosis is far and away the most frequent cause of pneumothorax, not always a manifest tuberculosis and particularly not a tuberculosis of an advanced degree with large cavity formation, etc., as you might at first think. When a lung has become so infiltrated that cavity formation exists, it is almost invariably the case that adhesions obliterate the pleural cavity at this particular spot, so that perforation into the free pleural cavity becomes impossible. On the other hand, it is the rather early case

that is likely to perforate, and particularly those cases in which there is fairly rapid caseation. A small and inconspicuous but rapidly developing focus is, in my experience, the most frequent cause of pneumothorax when this is due to tuberculosis.

In this patient there is absolutely no history of a pre-existing tuberculosis nor of any symptoms which might be legitimately interpreted as pointing toward tuberculosis. The patient had had no cough, no chills, no night-sweats, no loss of weight or strength, no anorexia, and had been about and attending to his work as usual. His digestion, sleep, and other bodily functions were perfectly normal so far as could be elicited by the most painstaking questioning. He had been examined repeatedly for life insurance, and nothing abnormal had been detected on these occasions. Even granting that these examinations are frequently of the most perfunctory kind, still they must carry some weight. Trauma is, of course, excluded, in the sense that the patient had met with no trauma from without. One might have thought of the possibility of a tumor with a metastasis in the lung ulcerating through, and, indeed, one colleague was strongly of this opinion, basing his judgment upon the fact that the fluid remained strongly hemorrhagic for so long a time, this indicating to him that there was continued bleeding. There was, however, absolutely no evidence that I could elicit of anything of that sort. Remembering that metastases occur in the lung very frequently in hypernephromata, which may themselves remain quite latent, the urine was carefully examined and the kidney region palpated for evidence of tumors there, but nothing of this sort could be found. The heart was examined carefully for evidences of dilatation or valvular disease, with entirely negative results. It is, of course, entirely unnecessary to say that the dislocation of the heart made such an examination particularly difficult, but the patient had had no previous diseases, such as rheumatism, tonsillitis, or chorea, which might make us think especially of endocardial disease. The reason for thinking of endocardial disease is that in mitral lesions with dilatation of the right heart, especially if there has been decompensation with a dilated right auricle, a small embolus may be

carried into one lung and the rupture of a hemorrhagic infarct thus resulting may give rise to pneumothorax. We thought of the possibility of perforation of the pleura from the abdominal cavity through the diaphragm, but there was nothing which in any way could be considered as a carcinoma of the colon or stomach. The esophagus seemed normal so far as could be told by the passage of a sound and swallowing. There was no evidence of carcinoma or stricture or diverticulum, all of which have been known to produce a rupture into the pleural cavity. Cases of bronchiectasis with cavity have also been known to produce pneumothorax, but examination of the sound lung showed no evidence of bronchiectasis. The same applies, of course, to abscess or gangrene.

Certain conclusions may be drawn from examination of the aspirated fluid. Cultures of this remained sterile. Inoculation of the fluid into a guinea-pig did not lead to the development of tuberculosis in the animal. A differential count was worthless because of the large amount of blood. The fact that the fluid was sterile showed that it was not a case of so-called spontaneous pneumothorax due to some gas-forming bacillus, so we seemed rather at a standstill so far as the discovery of the exciting cause was concerned. I have already voiced my unwillingness to accept any of the cases of spontaneous rupture of a normal and healthy lung that have been reported. When one thinks of the enormous exertions that can be made, involving the greatest possible strain upon the lung without any rupture, it seems to me utterly inconceivable that a normal lung should rupture under any ordinary or even extraordinary exertion, and still less so, of course, while at rest, despite the cases that have been reported as examples of this occurrence. It seems to me that in every such instance there must have been a small emphysematous bleb or a small focus of tuberculosis. It is notorious how difficult it is, particularly when the focus is deep seated, to determine the existence of tuberculosis, and this applies especially to such a case as this, where the patient has not been examined until after the development of the pneumothorax. What seems to me a much more probable suggestion

is that there are in these cases, where no active tuberculosis can be determined, some old adhesions in the vicinity of the apex, with perhaps the development of ectatic veins or a miliary aneurysm to account for the hemorrhage. It is quite conceivable that an old focus of tuberculosis at the surface of the lung leads to the formation of an adhesion, and that as the result of some awkward motion the adhesion may tear away a bit of the lung tissue and at the same time some of the vessels may bleed freely.

Our patient gave a definite tuberculin reaction on the subcutaneous injection of old tuberculin a couple of months after the onset of the disease. He at no time had any sputum, and in all the months that I have had him under observation since that time I have never been able to obtain any sputum spontaneously or after the administration of potassium iodid for many days. Now a tuberculin reaction cannot, I think, be by itself regarded as conclusive proof that the focus was due to tuberculosis. Bearing in mind the fact that we have failed to discover anything else, and also failed to determine clinically the tuberculosis in any other way but by the use of tuberculin, I think the most probable supposition is that we actually had a clinically obsolescent focus of tuberculosis which ruptured somewhat in the fashion that I have pictured to you. With the dismissal of the patient from the hospital after a seventy-two-day stay, the first chapter in his disease may be regarded as closed, and the second, for which I am now showing him to you, may be considered as beginning.

Let us now look over the patient and see what his condition is at the present time. Under forced feeding he has gained considerably in weight, although he is far from strong. His blood count and hemoglobin are now approximately normal and the white count absolutely so. He has not yet been able to resume business, although I think he really would be able to do so were it not for the haunting fear he has of his condition returning, and which fear, as I have before stated to you, is one of his leading symptoms.

It is the examination of the chest which interests us most

particularly. As we stand the patient up before us we will see that he has a very distinct thoracic asymmetry. The left side is distinctly drawn in at the lower portion and the respiratory excursion on that side is greatly lessened. As we turn him around with his back to us we see a very definite lateral curvature of the vertebræ. On asking him to take a deep breath the difference in respiratory excursion of the two sides is accentuated, the right side breathing very deeply, the left very indefinitely. Percussion shows a dulness over the entire lower portion of the left lung most marked posteriorly and in the axilla, and to a less extent in front. This dulness is so great as to be almost flatness, but close attention will convince you that there is a little bit of resonance left. The lung boundaries on the left side are absolutely immovable. On auscultation the breath sounds on the right side are a little exaggerated, otherwise normal. On the left they are diminished over the entire left lung, most markedly over the portion where we elicited dulness on percussion. The apex-beat is not to be found. The maximum precordial pulsation is to be found over the lower portion of the sternum, and just to the right of it in the fifth and sixth interspaces, and it is evident that the heart has been anchored in that position. The maximum intensity of the first sound is heard over the lower portion of the sternum in about the middle line. The sounds are clear, but the second pulmonic, or at least what we take to be the second pulmonic, is very plainly accentuated. The abdominal viscera show nothing abnormal, nor does the urine.

Now what is the explanation of this condition? Let us stop a moment and think what has happened. An individual substantially normal except for some relatively trifling focus in the lungs of one sort or the other, probably tuberculous, had a pain, coming on after a slight exertion, and immediately developed a pneumothorax with a copious hemorrhage. The shock of the pneumothorax, and particularly the hemorrhage, was so great as to almost cause death. Owing to the fact that the left side of the chest was put out of commission so far as breathing was concerned, the patient lay on his left side constantly, and it was

a very long time, at least not until he was ready to leave the hospital, before he could lie either on his back or on the right

Fig. 190.—Case of hemopneumothorax, taken several months after disappearance of effusion. Showing thickening of the pleura and compression of lung. (N. B.: The patient lay always on the left side.) Note the scoliosis and dislocation of the heart which has become fixed in that position.

side, and then only with great difficulty. He still has difficulty in lying in any position, and especially in sleeping in any position other than on the left side. As a result of this the pleural

effusion which was poured out secondarily found the most dependent portion to be the axillary region, and in consequence the thickening of the pleura, which undoubtedly is quite considerable, occurred in that location. If our interpretation of the accompanying plate is correct (exhibiting *x*-ray plate), the dark shadow seen up and down the left chest is due to a thickened pleura in that position. We must bear in mind the great mobility of the fluid in pneumothorax, which is far greater than in a simple pleurisy, and I think this accounts for the fact that the fluid took that position and kept it long enough for the pleura to become materially thickened. The lung is evidently pressed backward and inward against the vertebral column. Apparently it has regained a little of its normal condition low down along the vertebral column. The shadow of the heart, as you see, is away over to the right; indeed, it is not possible to make out any heart shadow to the left of the vertebral column. What has evidently happened is that a good deal of connective tissue has been formed as the direct result of the pleuritis which was secondarily produced, and which may be, of course, tubercular, although our examination of the fluid itself and inoculation experiments were negative for bacilli. This exudate has become organized and has produced a typical condition of retraction of the left chest (Schrumpfung). It is probable that the entire mediastinum has become fixed in the position in which it found itself after the rupture of the lung, and it is likely that it will remain there, or substantially there, since in the intervening months there has been no perceptible change in this position. One might think that the development of this connective tissue would drag it back again toward the left side, but such does not seem to have been the case. The lateral curvature is, of course, due to the retraction of the lung on one side and the compensatory emphysema which has now developed in the right lung.

There is another point worthy of note. We can already find this accentuation of the sound over the base of the heart, which we believe to be the pulmonary. This is due to the resistance offered to the right heart by the obliteration of many

capillary tracts in the left lung as a result of its collapse and pressure on the arteries and veins, with probably some additional increased resistance in the right lung due to the compensatory emphysema. As a result of this the right ventricle is undergoing hypertrophy because of the increased pressure in the pulmonary arteries, thus causing the pulmonary valve to close with greater force.

Regarding the outlook in such cases as this, I regard it as favorable as far as life is concerned, and, as we have already stated, the patient is in fairly good condition. I regard it also as favorable as regards a further development of the tuberculosis. Assuming that our diagnosis of tuberculosis is correct, this applies with equal force, since under these conditions with the left lung collapsed there is not, in my judgment, great liability of a lighting up of the process. On the other hand, it is not probable that there will be a very great deal of improvement in the affected lung, and I take it that there will be considerably more hypertrophy developed in the right ventricle.

I want to call your particular attention to this because of the great similarity in one respect of a case like this to a case of high-grade scoliosis. In Saxony, where such cases are so very common, it used to be an old observation, almost proverbial, that the normal end of a scoliosis patient when this was of high degree came as the result of a broken-down right heart, due to the obstruction in the pulmonary capillaries by compression in one lung and compensatory emphysema in the other. Romberg has laid great stress upon this, and I have had abundant opportunities of verifying these observations, which I think are not sufficiently well known. I should not be greatly surprised if this patient ultimately succumbed to a broken-down right heart, although at the present time there is no evidence of this. At any rate, we can say with certainty that the right heart is working against a very definite obstacle.

Treatment.—It is not my intention to discuss the treatment of pneumothorax in general, but merely to tell you what was done in this case. The treatment seems to have been followed by good results. My reason for aspirating the patient in the

first place was because it seemed to me that his condition was so serious as to make this procedure advisable. On the other hand, it was not certain that there was not some hemorrhage going on all the time for the first few days, so I felt that it was desirable to aspirate frequently and in moderate quantities, from ½ to 1 liter at a time, rather than to attempt to empty the pleural cavity completely at one sitting. I think it is considered good practice, and it is at least my practice, to do nothing to the ordinary case of pneumothorax which develops in the course of a tuberculosis. The reasons which prompt this method of treatment are the same which govern us in the production of artificial pneumothorax, namely, that the lung is benefited by the rest. The exceptions are in those cases where the intrathoracic pressure is so great as to be very serious and in those cases where a pleuritic exudate, whether serous or hemorrhagic, accumulates to such an extent as to render aspiration advisable for the removal of the fluid. Of course, if a frankly purulent exudate develops, it should be treated as any other empyema. A further reason for aspiration in these cases is to be found in the pressure of the fluid causing collapse of the lung, and subsequently, when adhesions form, preventing its proper expansion. In our patient, after his lung seemed to me to be safely healed, I had him practice blowing water from one Woulff bottle to another, as in the well-known after-treatment of empyema.

The *x*-ray plate of his chest shows the extent to which the lung is collapsed, or rather does not show the left lung at all, but shows the result in the shape of a deformity produced by the collapsed lung and the thickened pleura. Of course, it goes without saying that, believing this case to be tubercular, I have put the patient upon forced feeding and outdoor life as much as practicable, and the results have been very satisfactory. Some of you may disagree with me, and think that the evidence pointing toward an old tuberculous lesion in the lung was insufficient, and may prefer to call this case one of rupture of a normal lung, but I have never been able to convince myself that this latter accident actually occurs. Certainly, from the standpoint of treatment, it seems wiser to consider the case as due to a low-grade tuberculosis.

CLINIC OF DR. ARTHUR F. BEIFELD

Cook County Hospital

PERNICIOUS ANEMIA

Morawitz's Classification of the Anemias. Diagnostic Routine in Pernicious Anemia. Differential Diagnosis. Relation of Syphilis to the Hemolytic Anemias. Treatment.

In an earlier clinic devoted to pernicious anemia (Vol. II, No. 2, page 395) I considered the differential diagnosis of a case from the point of view of its most striking characteristic, namely, the presence in the circulating blood of an enormous number of nucleated red blood-corpuscles. At that time I postponed for a later discussion certain other aspects of the differential diagnosis, and in particular that based upon etiologic considerations.

This morning I purpose taking up this very important phase of pernicious anemia. This is not a matter of diagnostic *finesse*, but of gaining an insight into certain fundamentals without which satisfactory work in this field is impossible. Pernicious anemia is a difficult subject, a fact due not to the inability of the student to grasp our available knowledge of the matter, but to the existence of two other conditions—first, the obscurity of the cause or causes of the disease, and, second, the disagreement among hematologists as to what permits of a diagnosis of the condition with the criteria at present available.

History, Physical and Laboratory Findings.—The case before us—a man of thirty-two, a bookkeeper by occupation—is one especially well adapted to the opening of this discussion. For the following reasons I consider it an undoubted example of pernicious anemia:

1. The subjective disturbances of the patient, which are physical weakness, dyspnea on slight exertion, cardiac palpita-

tion, dizziness, dyspeptic symptoms, and bleeding from the nose and gums.

2. The history of two previous similar attacks, one eighteen and the other six months ago.

3. The physical findings, which include a typical straw yellow color of the skin, with pale mucous membranes; a well-preserved adipose tissue; a hemorrhagic diathesis, manifested by bleeding gums, epistaxis, "cherry spots" in both retinæ and petechial spots on the palate; a moderately dilated heart, exhibiting a loud non-valvular murmur over the entire precordium, but heard most distinctly over the base to the left of the sternum and only faintly transmitted to the axilla; tachycardia, the pulse-rate averaging over 90 beats to the minute; diminished arterial tension (systolic 105, diastolic 80); a slightly enlarged spleen as shown both by percussion and palpation; tingling in the hands and feet; and well-marked ankle edema.

4. A dark urine, albuminuria without cylindruria, the presence of a decided Ehrlich aldehyd reaction for urobilinogen, and the absence of free hydrochloric acid in the gastric juice.

5. Characteristic hematologic findings, as follows:

Erythrocytes, 990,000.

Poikilocytosis and anisocytosis marked. Of the cells varying from the normal in size, megalocytes and gigantocytes considerably outnumber microcytes. The hemoglobin content of the individual cell is good despite the high grade of anemia present. Nucleation, basophilic granulation, and polychromatophilia absent.

Hemoglobin, 25 per cent. (Sahli corrected. See Vol. II, No. 5, p. 977.)

Color-index, 1.3.

Leukocytes, 2600, distributed as follows:

	Per Cent.
Polynuclear neutrophils	44
Lymphocytes	53
Large mononuclears and transitionals	0.5
Polynuclear eosinophils	0.5
Neutrophilic myelocytes	0.5
Large mononuclear cells with homogeneous, basophilic cytoplasm—probably pathologic lymphocytes	1.5

Blood-platelets much reduced.

This was the blood-picture when the patient was admitted to the hospital. Now, after the lapse of nearly six weeks, con-

ditions have changed considerably for the better, whether as the result of treatment or coincident with the appearance of a remission we shall consider later in the hour. If you will refer to a table which I gave you in the earlier clinic already mentioned (see page 411) you will note that the blood findings I have just read correspond very closely to what is there termed a period of greatest severity. Hand in hand with the improvement in the condition of the blood has gone an amelioration of the patient's subjective disturbances to the extent that he sleeps and eats well, experiences much less palpitation, can sit up in bed with but little vertigo, and is beginning to be interested in the time when he can get his clothes. The bleeding from the mucous surfaces has also ceased. In brief, there seems little doubt that the man is well on his way to a recovery from an attack—his third—the prognosis of which at the time of his entrance into the hospital looked very dubious.

We have to do, therefore, with a case in which the subjective symptoms, the history, and the physical and laboratory data point unmistakably to pernicious anemia. A doubt as to the correctness of the diagnosis may suggest itself to you, first, because of the age of the patient, and, second, because of the absence of nucleated red cells in the blood-smear. With respect to the first point, I agree that while thirty-two is considerably under the average age at which we observe the disease, it is not altogether rare; and, as to the second, the failure to find erythroblasts in a single specimen, or at certain very severe periods in the course, is, as I have already emphasized, of common occurrence (see Vol. II, No. 2, pages 403 and 411). The diagnosis of an aplastic anemia, as we shall have occasion to see at another time, is a difficult one to establish and is made all too frequently, to judge from autopsy statistics.

Classification of the Anemias in General.—Before proceeding to a further discussion of this case of apparent pernicious anemia I shall give you a classification of the anemias in general in order that you may more readily appreciate the position of pernicious anemia in this group of diseases, and at the same time learn the essential diagnostic criteria. No classification

can be regarded as entirely satisfactory until one can be built which shall be based upon a full knowledge of the etiology of certain obscure forms. The following grouping, arranged by Morawitz, is perhaps as logical as any from the point of view of our present knowledge:

Morawitz's Classification of the Anemias

I. Anemias due to increased blood destruction.
  1. Posthemorrhagic:
    (*a*) Acute,
    (*b*) Chronic.
  2. Hemolytic:
    (*a*) Due to hemolytic or methemoglobin-forming poisons (pyrodin, phenylhydrazin, nitrobenzol, potassium chlorate, etc.).
    (*b*) Due to Bothriocephalus latus and rarely to other helminths.
    (*c*) Of unknown origin.
      (1) Acute, generally with leukocytosis (rare).
      (2) Chronic (pernicious anemia—Addison-Biermer type).
    (*d*) In the puerperium, syphilis, malaria, and in rare cases of carcinoma.

II. Anemias due to deficient blood building.
  1. Myelopathic: Due to encroachment upon the bone-marrow by tumors, osteosclerosis, etc.
  2. Hypoplastic, for example:
    (*a*) In wasting diseases, inanition, and in anemias due to tumors of various organs.
    (*b*) Aplastic, or aregenerative (cause unknown).

III. Chlorosis.

IV. Anemias of Infancy.

A few words are necessary in explanation of this classification. In the first place, the term *hemolytic* is employed not in the sense that the hemoglobin of the erythrocytes escapes with abnormal ease from the corpuscular envelopes, as in certain forms of hemolytic icterus, but implies merely that there is taking place—within the body—an excessive destruction of the red blood-corpuscles, chiefly in the liver and spleen. This is evidenced clinically by the dark color of the urine, the frequent subicteric color of the skin and mucous membranes, and the greatly increased amounts of urobilin and urobilinogen in the urine; and at autopsy by a hemosiderosis of the organs, in particular of the liver and spleen.

The acute hemolytic anemia of unknown origin mentioned in the table is the *leukanemia* of Leube and Arneth. According to these observers, this type is characterized by certain of the features both of pernicious anemia and of myeloid leukemia—hence the name. Pernicious anemia is suggested by the embryonal type of erythrocytes present—megalocytes, megaloblasts—and leukemia by the increased white cell count and the numerous myelocytes. A more rigid criticism, however, has convinced most observers that the so-called leukanemia is not an entity, but includes a number of rather widely differing conditions, such as the acute leukemias, atypical pernicious anemias, anemia pseudoleukemica infantum, sepsis, etc. It is probable that many, if not most, of the cases described as leukanemia were, in reality, acute leukemias. Speaking for this are the apparent infectious nature of the process—fever, etc.—the increase in the number of leukocytes, the hemorrhagic diathesis, and the marked disturbance in the leukopoietic function (myelocytes, large mononuclear cells with basophilic non-granular cytoplasm); nor is the embryonal character of erythropoiesis a rare finding in the acute leukemias, as we have seen in another case. (See Vol. II, No. 4, pages 855 and 858.)

That form of *hemolytic anemia due to the Bothriocephalus latus* is of especial interest not only because it presents a hematologic picture indistinguishable from that of the idiopathic type, and because it is unquestionably curable if the cause be removed before too great damage has been done, but also because research into the nature of bothriocephalus anemia has thrown considerable light upon the etiology and pathogenesis of the Addisonian and other forms of hemolytic anemia.

In the *myelopathic anemias* there is a deficient building of blood owing to the encroachment upon the bone-marrow of processes which tend to strangle the activity of the marrow. Tumors may do this, as may also osteosclerosis, while in leukemia we often see the bones so thick as to leave only a narrow band of medulla. Anemia, naturally, will be the result of such conditions. The appearance in the blood of evidences of red and white cell regeneration—nucleated erythrocytes, myelo-

cytes—is assumed to be due to a primary stimulation of the marrow, comparable to nerve stimulation preceding nerve degeneration.

The *hypoplastic anemias* associated with wasting diseases, inanition, and tumors of various organs include a great many of the forms we are accustomed to catalogue as secondary anemias.

As to *aplastic* or *aregenerative anemia*, I plan to devote a considerable discussion to the subject in a later clinic. However, in view of the wide-spread misconception of what is meant by the term, I cannot forbear saying a few words at this time. As in the case of pernicious anemia, the cause is quite unknown. Further, it is held by many observers that aplastic anemia differs from the Addisonian type only in the complete absence of regenerative response on the part of the bone-marrow. *Nucleated red cells are constantly absent.* Remember this, however, the failure to find erythroblasts in a number of blood-smears taken from a case of pernicious anemia at one time, or even in smears made over a considerable period of time, by no means eliminates the possibility of a pernicious anemia. Nucleated erythrocytes are never found in great number in the latter except during the occasional blood crises, and they are frequently absent for considerable periods when the anemia is especially severe.

Other hematologists do not regard aplastic anemia as an aberrant form of the pernicious type. Morawitz, as you will observe in the table, calls it a hypoplastic anemia—*i. e.*, one due to a bone-marrow insufficiency. The characteristic signs of the hemolytic anemias, indeed, are wanting, and at autopsy we find in the long bones a yellow marrow instead of the red marrow characteristic of erythropoietic activity.

Chlorosis has been included in the table of anemias by Morawitz more by courtesy of custom than because it rightfully belongs there. As I have already indicated to you at great length (Chicago Clinics, March, 1917) Morawitz looks upon the anemia of chlorosis as only one symptom of a larger, as yet little understood, disease process. I shall not repeat the various theories as to the nature of chlorosis which I discussed in that

earlier clinic. Though there are a great many things speaking for the correctness of Morawitz's view in this matter, you must not leave with the impression that chlorosis has definitely been displaced from its position as a primary anemia.

Finally, regarding the *anemia pseudolcukemica infantum* (von Jaksch), there is also considerable diversity of opinion as to where it belongs. This is due to two main causes: First, the fact that it is more a disease complex than a disease entity, having been made to include anemias of varying types; and, second, because, though it bears a number of the earmarks of the hemolytic anemias, yet it differs from these in certain important details.

Diagnostic Routine in Pernicious Anemia.—You should now be in a better position to orient yourselves in the diagnosis of pernicious anemia. For example, you see that my use of the term "pernicious anemia" as applied to the patient before us was not justified on the basis solely of the information I have given you. It is a hemolytic anemia, it is true, but not necessarily of the chronic form of unknown origin called the Addison-Biermer type. In other words, the diagnostic routine entails first the classification of the condition before you as an anemia; second, correlating the anamnestic and laboratory data at hand, the type of anemia present. We have reached this point in the patient we are studying—*i. e.*, we have to do with a hemolytic anemia. Before one is justified in making a definitive diagnosis of pernicious anemia one must exclude beyond all reasonable doubt all of the other forms classed in the table as hemolytic anemias. As I said at the beginning of this clinic, this is more than a matter of diagnostic *finesse*, for upon a careful study of this point may depend the very life of the patient. Pernicious anemia differs from the other hemolytic anemias only in the absence of a, as yet, discovered cause, while those forms with a known etiology are amenable to cure if the cause be recognized early enough and removed.

In our patient we can at once rule out the acute form of unknown cause—the so-called leukanemia. The history, furthermore, warrants us in eliminating the subtype due to hemolytic

or methemoglobin-forming poisons. Repeated examinations of the stool, employing also the Yaoita[1] antiformin method of rendering the ova of the Bothriocephalus latus more accessible, have ruled out this form to our satisfaction. Nor were ova of other helminths which rarely produce the hemolytic type of anemia discovered.

In regard to the severe forms of anemia due to intestinal parasites, I wish to emphasize one point which your texts generally fail to touch upon, namely, the behavior of the eosinophils. The latter progressively diminish as the anemia becomes more marked, so that as the blood-picture approaches the pernicious in character an eosinophilia is no longer present, and one of the most valuable diagnostic features of the parasitic etiology is, unfortunately, gone.[2]

We come finally to a consideration of the fourth subdivision of the hemolytic anemias—those associated with the puerperium, syphilis, malaria, and rarely with cases of carcinoma. There is more or less general agreement among hematologists that the first two conditions mentioned may produce a clinical and hematologic picture indistinguishable from that of the true Addisonian type. A discoverable etiology which, if removed early enough, permits of complete recovery without recurrence is the sole differentiating feature. However, the syphilitic and the puerperal forms are surely not very common if one may judge from the few recorded cases.

Differential Diagnosis.—As to the relationship of severe chronic malaria and isolated carcinomas to the pernicious type of anemia, there is a considerably greater difference of opinion. In connection with this point we may very appropriately consider the differential diagnosis of pernicious anemia. You will recall that we have already pointed out that the Addisonian anemia can be separated from that due to the bothriocephalus, to syphilis, and to the puerperium only by the absence of a discoverable etiology. Is this true of those other conditions pro-

[1] Deutsch. med. Woch., 1912, xxxviii, 1540.

[2] Beifeld and Barnes: Severe Anemia with Eosinophilia, J. H. H. B., xxvii, 1916, 181.

ducing an anemia closely resembling the pernicious? This question opens up one of the most discussed points in connection with the subject. On the one side we may mention Naegeli, who maintains that pernicious anemia and the bothriocephalus, syphilitic, and puerperal anemias produce a blood-picture of such characteristic detail as to enable one to differentiate them in practically every case from all other anemias resembling them. Naegeli feels competent, in other words, on the basis of the blood findings alone—the embryonal type of erythropoiesis (megalocytes, megaloblasts predominating if only a few nucleated red cells are present), the high color index, the leukopenia with a relative lymphocytosis, the blood-platelet diminution, etc.—to render a positive diagnosis of the Addisonian type of anemia.

Opposed to this point of view of Naegeli are among others Grawitz, who takes the position that, while the average picture of true pernicious anemia is characteristic, it may be produced by conditions having to all appearances nothing in common with Addisonian anemia. In Grawitz's opinion—and this is strongly maintained also by Krehl—the so-called pernicious blood-picture may be exhibited not only by the idiopathic form and by syphilis, Bothriocephalus latus, and the puerperium, but also by a variety of other conditions, such as carcinoma of the stomach, gastric ulcer, hepatic affections, chronic losses of blood, diseases of the bone-marrow, etc. In their opinion the effort to find certain diagnostic blood changes—high color index, megaloblasts, etc.—belonging to true pernicious anemia alone has not proved successful. Such changes are to be regarded merely as evidence of an especially stormy regeneration of the erythrocytes, which, because of the inadequacy of normal sources of new red cells, assumes the type observed in the embryo.

My own experience has inclined me more to the view of Naegeli than to that of Grawitz and Krehl. In other words, I have in almost every doubtful case been able to find in the blood-picture some feature confirming or eliminating the diagnosis of a pernicious anemia, and an autopsy when obtained has

borne out the differentiation. Also it is obvious that the history and physical and laboratory findings in the particular case help very materially in pointing toward the correct diagnosis. For example, *carcinosis of the bone-marrow* may produce a picture in most respects closely resembling that of pernicious anemia. Yet there are such sharp differences as a *persistent* flooding of the circulating blood with nucleated red corpuscles in large numbers—a condition never observed in the pernicious type—a considerable leukocytosis, and the presence of numerous myelocytes.

In the *anemias of infancy*, particularly in the von Jaksch type, there is an outspoken embryonal character to the erythropoiesis. Here the marked leukocytosis, the myelocytosis, and the large number of nucleated erythrocytes are distinguishing features, while the enormous spleen and the large liver are additional differences. *Chronic malaria* which has resulted in an anemia of high degree exhibits besides a large hard spleen, as a rule, a leukocytosis and a marked and persistent erythroblastemia.

Carcinoma of the stomach is one of the most fruitful sources of confusion for various reasons. In the first place, carcinoma may, without attracting attention to the stomach, cause an anemia of the severest grade. Second, both carcinoma and pernicious anemia are associated generally with an achylia. Again, cases are on record which must be catalogued as examples of unquestioned Addisonian anemia in which a small non-ulcerating gastric cancer was discovered at autopsy. This is exceptional, however, and must be regarded as an accidental association, for the average stomach cancer produces a blood-picture which has few of the earmarks of the pernicious, being characterized by an anemia of the secondary type, *i. e.*, absence of embryonal features, low color index, leukocytosis, etc.

If our time permitted I might continue with the differential diagnosis, and point out how the other conditions commonly mistaken for pernicious anemia—*ulcerative endocarditis, cirrhosis of the liver, chronic nephritis, Addison's disease, chronic gastro-intestinal disturbances* leading to a high degree of inanition, etc.—

may all be ruled out not only by the anamnestic data and the results of the physical and laboratory examinations, but also by distinguishing hematologic findings.

Diagnosis of the Case Presented.—I must conclude this interesting phase of the problem, however, and return to our patient whom we have long neglected. We have decided that we have to do with a hemolytic anemia not falling under subdivisions (*a*) and (*b*) of Morawitz's classification. Further, I have given it as my opinion that malaria and carcinoma produce blood-pictures which can be separated more or less sharply from that of the Addisonian form. There is left to consider, therefore, only syphilis, before a diagnosis of true pernicious anemia is permissible.

And, as a matter of fact, this young man has a clear case of lues. The diagnosis rests not only upon the history of chancre, cutaneous lesions, mucous patches, headache, bone pains, etc., but also upon a strongly positive Wassermann reaction in the serum. (The spinal fluid shows negative Wassermann and globulin reactions and a normal cell count.) The infection is of ten years' standing, and, though previously recognized, has never been efficiently treated. At the present time, however, the only sign of the activity of the infection is the fixation of complement.

Relation of Syphilis to Hemolytic Anemias.—We have before us, therefore, the characteristic findings of a hemolytic anemia and also one of the recognized causes of such an anemia. Does this establish a positive etiologic relationship? By no means. In view of the great prevalence of syphilis, it is not at all surprising that in many cases of pernicious anemia there should be a history of antecedent lues or even more positive evidence, such as a Wassermann reaction. Syphilis can be accepted as the cause of the particular case of hemolytic anemia only if treatment effects a permanent cure. Remissions, as you know, are of the very essence of the disease itself, and cannot be accepted as proof of the efficacy of treatment. However, there are a few cases on record in which, with a luetic infection, a permanent cure of the hemolytic anemia has been effected by a proper treatment of the syphilis.

Treatment.—The indication for treatment in our case, therefore, is very clear whether the syphilis is responsible for the trouble or not. As has already been said, the man has never had the benefit of a thorough course of antiluetic therapy either before or after the onset of the anemia. As soon as the diagnosis of syphilis was established by us he was given mercurial rubs twice a day for a week, after which neosalvarsan in increasing doses was introduced intravenously once a week. He received his fifth injection—the full dosage, 0.9 gm.—yesterday. As the urine has given no evidence of renal irritation, the mercurial inunctions have been continued between the neosalvarsan injections, though after the first week once a day only. As you see, we are pushing the treatment as rapidly as safety will permit.

I have told you that the patient has shown a considerable regression of his subjective symptoms, and, if you will compare the blood findings now with those I gave you as being present when the man entered the hospital, you will observe in this regard also a marked improvement. The following blood-picture was obtained two days ago:

Erythrocytes, 1,800,000.

Poikilocytosis and anisocytosis considerable; megalocytes numerous. Basophilic granulation and polychromatophilia fairly well marked; 2 megaloblasts per hundred leukocytes counted.

Hemoglobin, 40 (Sahli corrected).

Color-index, 1.1.

Leukocytes, 4200, distributed as follows:	Per Cent.
Polynuclear neutrophils	54
Lymphocytes	42
Large mononuclear and transitionals	2
Polynuclear eosinophils	1
Neutrophilic myelocytes	1

There is nothing specific in this change for the better, only the usual signs of an improvement. The picture is, of course, still entirely typical of the hemolytic anemias. A period of observation extending over several years and more will be necessary before a final conclusion can be reached as to whether this case is one of the Addisonian type or is a hemolytic anemia resembling the pernicious form, but due to syphilis.

NOTE.—Six months after the presentation of the patient in clinic his condition is practically normal. His subjective disturbances have entirely disappeared, he has lost his lemon-yellow color, and appears, in fact, in perfect health. Of the earlier findings there remain a barely perceptible systolic apical blow, a slight tachycardia (82 beats to the minute in the recumbent position), moderate hypotension (110 mm. systolic), achlorhydria, and, of course, the relics of the old retinal hemorrhages. As for the blood, the hemoglobin percentage is 85 and the red cell count 4,100,000, the color-index, therefore, remaining slightly above 1. Megalocytes are to be seen here and there, and, after a search of a number of slides, one megaloblast was found. The hemolytic character of the condition, in other words, can still be diagnosticated, and the definitive diagnosis must be further postponed.

CLINIC OF DR. B. C. CORBUS

RUSH MEDICAL COLLEGE, CENTRAL FREE DISPENSARY

GONORRHEAL ARTHRITIS

Frequency of Gonorrhea as a Focal Infection. Osler's Classification of Gonorrheal Arthritis. Pathologic Changes in the Joints. Differential Diagnosis. Importance of the Complement Fixation Test in Systemic Gonococcus Infections. Treatment with Special Reference to the Use of Antigonococcic Serum.

CONSIDERING the great publicity that is being given to focal infections I have thought it might be well to call your attention to a class of infections that are too little recognized. When we stop to consider that gonorrhea is the most prevalent of all infectious diseases and leads to chronicity, it is surprising that we do not have more gonorrheal focal infections with their accompanying arthritis.

Gonorrheal arthritis is the most serious and damaging of all the complications of gonorrhea, occurring, according to most authorities, in from 2 to 5 per cent. of cases. It occurs most frequently in men, and, as a rule, comes on during the acute stage, but may occur at any time while the gonococcus is in the urethral canal or its appendages.

Dr. E. J. Angle cites in his excellent monograph a case of ophthalmia neonatorum occurring forty-eight hours after birth, followed two weeks later by an arthritis involving the left wrist and right knee; also a second case infected in a similar way affecting both wrists.

From what I have said it is evident that gonorrheal arthritis is not limited in its origin to the urinary and genital tracts.

Recently I examined a patient's wife who had her tonsils removed for rheumatism, little suspecting that her case was

one of gonorrheal arthritis. Gonococci were demonstrated in the urethra, however, and the diagnosis was confirmed by a positive complement fixation test.

One must always bear in mind that the different forms of arthritis are due to focal infections most frequently traced to the tonsil and teeth, occurring, however, much more frequently as a result of gonorrhea than is generally supposed. Often by a little tact and discretion the physician can determine an unsuspected focal infection and the patient still remain in possession of his perfectly good teeth and tonsils.

History

Name: M. S., male. Date, 11, 16, 1916.
Occupation: Clerk. Age: Twenty-one years.
First urethritis, July 4, 1916. Treatment: Irrigations of kalium permanganate.
Complications: Prostatic abscess one month later, followed three weeks later by arthritis in right and left ankles.
Diagnosis: Subacute gonorrheal arthritis.
Chronic gonorrheal prostatitis.
Urethritis totalis.
Discharge: Gonococcus.
Urination: Night: Occasionally. Day: 4 to 6 times. Pain: None. Blood: None.
Urine glasses: I. Turbid. II. Turbid. III. Turbid after massage. Large quantity of pus present.
Sexual symptoms negative.
Stricture: Negative.
Prostate: Large, boggy prostate.
Pus easily expressed.
Seminal vesicles: Negative.
Testicles: Negative.

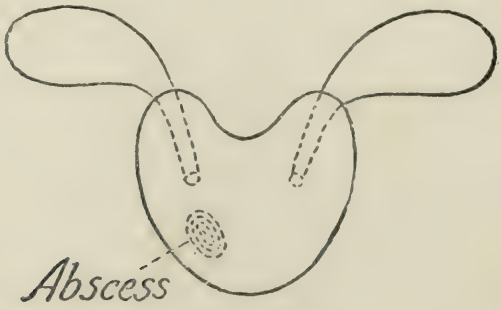


Fig. 191.

The man stated that during the third week of the disease a prostatic abscess developed. He was unable to void urine and had to be catheterized; he was in bed for two weeks with a high fever and some delirium. Three weeks later arthritic symptoms began to appear. At present the arthritis is of three months' duration. The patient is unable to walk without a cane; his general physical condition is good.

Complement fixation test, positive.

Leukocyte count, 8100.

Temperature and pulse, normal.

x-Ray shows no impairment of joint structures.

From the history of this case we can readily see that the following are to be considered:

I. The primary infection.

II. The spread in the genito-urinary organs by direct continuity.

III. Systemic gonococcus infection.

This patient became infected in July, 1916. Irrigations were immediately instituted, and one month later a prostatic abscess developed, showing the spread of the infection in the genito-urinary organs by direct continuity. Three weeks later arthritis appeared in both ankles as a result of systemic gonococcus infection.

Classification of gonorrheal arthritis, according to Osler, is as follows:

(*a*) *Arthralgic*, in which there are wandering pains about the joints without redness or swelling. These persist for a long time.

(*b*) *Polyarthritic*, in which several joints become affected. The fever is slight; the local inflammation may fix itself in one joint, but more commonly several become swollen and tender. In this form cerebral and cardiac complications may occur.

(*c*) *Acute gonococcus arthritis*, in which a joint, usually the knee, becomes suddenly involved. The pain is severe, the swelling extensive, due chiefly to peri-articular edema. The general fever is not at all proportionate to the intensity of the local signs. The exudate usually resolves, though suppuration occasionally supervenes.

(*d*) *Chronic Hydrarthrosis.*—This is usually mono-articular and is particularly apt to involve the knee. It comes on often without pain, redness, or swelling. Formation of pus is rare.

(*e*) *Bursal and Synovial Form.*—This attacks chiefly the tendons and their sheaths, the bursæ, and the periosteum. The articulations may not be affected. The bursæ of the patella, the olecranon, and the tendo achillis are most apt to be involved.

(*f*) *Septicemic form*, in which, with an acute arthritis, the gonococci invade the blood, and the picture is that of an intense septicopyemia, usually with endocarditis.

(*g*) *The Painful Heel of Gonorrhea.*—This is a remarkable form of pododynia due to local periosteal thickening and exostosis on the os calcis, causing pain and great disability. Baer has demonstrated the gonococcus in the periosteal lesion.

Complications.—Iritis is not infrequent and may occur with successive attacks. Endocarditis, pericarditis, and pleurisy may occur.

The case here reported can be classed under the heading (*c*), except that at the time the patient came under treatment the acute symptoms had subsided, but pain was severe and the swelling marked in the right ankle.

Pathology.—According to Angle, Koenig recognizes four varieties: (1) Arthritis with serous effusion; (2) arthritis with serofibrinous and catarrhal exudate; (3) empyema of the joint; (4) phlegmon of the joint.

In the first variety there is little structural alteration in the synovial membrane and the joint is more or less distended with a clear, serous fluid.

In the second form the synovial membrane is the seat of more decided infiltration and the fluid contains flocculent masses of fibrin.

In the suppurative variety, which is more severe though less common than the preceding, the joint cavity is filled with pus. According to Osler, mixed infection is rare.

The fourth variety—*i. e.*, phlegmonous and, fortunately, the rarest—works the greatest ravages on the joint and surrounding

structures and is, therefore, followed by the greatest functional disturbance.

Diagnosis.—Gonorrheal arthritis must be differentiated from arthritis deformans, acute and chronic rheumatism; from tuberculous and syphilitic diseases of the joints.

There should be no great difficulty in recognizing the existence of gonorrheal rheumatism, as it always occurs in connection with or following an acute urethral inflammation or an exacerbation of chronic infection; consequently one of the most important steps in the diagnosis is the history. If it points to a recent attack of gonorrhea, in a large proportion of cases foci containing gonococci can be demonstrated in the prostate gland or seminal vesicles.

In establishing and confirming the diagnosis of gonorrheal arthritis, microscopic examination is made for the gonococcus of all secretions from the genital tract. Frequently these contain many extraneous organisms, and Gram-staining, after the method described in the March Clinic, may be necessary to clear up the diagnosis.

The symptom-complex of gonorrheal arthritis is quite distinctive from that of acute rheumatism. In gonorrheal arthritis the fever is not so high; there is less prostration and sweating and an absence of the sudden subsidence in one joint and the equally sudden metastasis to another, as seen in rheumatism.

The pain symptom is quite characteristic. As long as the joint is at rest little if any complaint is made of pain, and the patient is fairly comfortable. I may add that there is no leukocytosis, as is found in general septic infections.

Chronic gonorrheal arthritis is far more difficult of diagnosis. There is no question in my mind that no inconsiderable percentage of the cases diagnosed as chronic rheumatism, neuritis, and neuralgia attributed to focal infections of the sinuses, tonsils, and teeth is due to the gonococcus.

Further, and most important of all, is the application of the complement fixation test for gonorrhea. This test is most valuable in systemic gonococcus infections and if properly performed the result will be positive in 100 per cent. of such cases.

When the diagnosis is in doubt the complement fixation test should always be employed if there is a history of gonococcic infection, no matter how remote. Frequently the injection of large doses of gonococcic vaccine will be followed by a febrile reaction with pain and local tenderness in the focal points of infection, *i. e.*, vesicle, prostate, or joints.

The technic is essentially the same as that of other complement fixation tests, but as different strains of gonococci differ considerably, and the serum of the patient infected with one strain will fix complement only in the presence of an antigen of the same or a very closely related strain, it is necessary to use in the test a polyvalent antigen. This should consist of an extract of from ten to fifteen different strains. Recently Warden has shown that the results are much more reliable when an antigen containing the fatty elements of several different strains of the gonococci is used.

Treatment of Gonorrheal Arthritis.—By consulting the accompanying history it will be seen that this patient has, in addition to the arthritis, a chronic gonorrheal urethritis totalis, and, therefore, before we can expect to cause resolution in the joint it will be necessary to eliminate the focal infection and its source—*i. e.*, the disease of the prostate and urethra—just as is attempted in other forms of arthritis by extracting teeth and removing tonsils, etc.

Prophylaxis.—In a previous clinic the care and management of simple acute and chronic gonorrheal urethritis was described in detail, attention being called to the seriousness of complications following posterior involvement. If every physician would constantly bear this in mind much could be avoided in the way of future complications of this form of infection.

It has been pointed out by numerous observers that a small external urethral orifice, permitting of poor drainage, is often followed by posterior involvement; also the practice of crowding the foreskin with cotton over the meatus leads to the same result. By all means in every case permit free exit of the pus, even if a meatotomy has to be performed.

Management of Urethral Infections.—The treatment outlined

in a previous clinic under Posterior Involvement must be continued here. Systematic and regular massage of the prostate must be carefully carried out if we are to remove the focal infection.

General Treatment.—The continued use of medicines for this and that symptom should be condemned. Our aim should be to get rid of the focal infection as soon as possible and to control complications. Medicines prescribed for rheumatic conditions have little if any effect. Salol and urotropin are valueless. At times sedatives and hypnotics may be useful. Later, when there is much debility and emaciation, iron, quinin, and strychnin, or syrup of iodid of iron may be given. However, nothing is more stimulating than plenty of fresh air and good food. A change of climate or a trip to some of the southern warm bath resorts is always beneficial.

Specific Treatment.—In 1907 John C. Torrey, of the Loomis Research Laboratory of New York, read before the American Congress of Physicians and Surgeons in Washington, D. C., an original communication describing an antigonococcic serum prepared by injecting dead and virulent gonococci into uncastrated rams. The indications for the employment of the serum were the following:

1. Those infections arising by direct extension; that is, in the prostate, bladder, and epididymis in the male, and in the uterus and fallopian tube in the female.

2. Those infections arising by extension through the lymphatics or the circulatory system. These include arthritis, iritis, endocarditis, pleuritis, and meningitis.

The dose, as recommended at that time, was 2 c.c., given every three or four days, at the discretion of the physician.

Previous to 1911 I had repeatedly given as large a dose as from 6 to 12 c.c. in two days in cases of acute epididymitis, with surprising results. In 1911 I had a beautiful illustration of the efficacy of this serum in a case of gonorrheal rheumatism accompanying prostatic infection. At this time 16 c.c. were given in three days. A complete cure was obtained in two weeks.

Stimulated by the work of Theobald Smith and Rufus Cole with pneumococcic serum, and of George Weaver with antistreptococcic serum, in September, 1912, I made a careful survey of my cases already treated and determined to adopt a larger dose. As the complement fixation test for gonorrhea is a reliable guide for systemic invasion, I selected this method of diagnosis as a key to the administration of the serum.

I have administered the antigonococcic serum in many cases; frequently the serum sickness is alarming, but with the judicious injection of adrenalin (1 : 1000) the symptoms are easily controlled. The following is given as a guide to the administration of the serum in gonorrheal arthritis:

1. The complement fixation test is a reliable guide in the administration of the serum. When the test is negative the serum should not be used.

2. The intensity of the positive complement fixation test is a satisfactory indication as to whether or not the serum will be efficient, as the efficiency and intensity are in direct proportion.

3. The amount injected should be at least 36 to 45 c.c., administered intramuscularly, from 12 to 15 c.c. a day for three days.

4. Serum sickness, if distressing, should not be alarming.

5. A negative complement fixation test after two or three months shows a complete cure.

Local Treatment.—So rapid was the improvement following urethral treatment, accompanied by prostatic massage and injections of antigonococcic serum, that it was not necessary to put this patient to bed. However, in the more severe cases it may be necessary to put the patients to bed and manage them somewhat after the following method:

The joint should be immobilized with a light splint. The affected limb should not be used until the acute inflammation has subsided, however mild it may be. Applications may be either hot or cold, as the patient finds most soothing. The following can be used with benefit: Compresses moistened with a hot solution of aluminum acetate, 2 to 5 per cent., or with

lead-water and opium solution. The compresses should be quite thick and covered with a dry towel, and over this rubber tissue or oiled silk.

When the inflammation in the joint runs high, or where the symptoms point to suppuration, the ice-bag is unquestionably the best application.

Later on, as the acute symptoms subside, the treatment should be directed toward absorption of the fluid in and around the joint. To stimulate absorption many measures are recommended, among which are light touches of the Paquelin cautery, blisters, iodin, iodin-petrogen, ichthyol, dilute mercurial ointment, and the rubber bandage.

Hyperemic Treatment.—This is applied by means of the rubber bandage, moderately tight, in several layers around the limb, reasonably well above the joint. It will soon be noticed that the superficial veins swell, that the skin becomes red, and that the limb becomes larger and edematous, and at the same time the skin becomes warmer to the touch. Pain rapidly ceases and stiffness in the joint gradually becomes less.

If the pain increases, the bandage is too tight. Everything depends upon securing the proper degree of hyperemia. The arterial circulation must be unimpeded and the return venous flow checked, but not entirely suspended. Previous painting of the joints with tincture of iodin will increase the hyperemia.

In the beginning of treatment it is best to leave the bandage *in situ* for only a few hours, but the duration is rapidly lengthened until the periods are ten hours during the day and ten hours at night. The benefits are threefold: (*a*) Relief of pain; (*b*) destruction of bacteria, depending upon increased quantity of serum and leukocytes flowing to the diseased joint; (*c*) the absorption of fluid and liquefaction of the fibrinous products in the joint.

To summarize:

1. The customary internal and local treatment has little influence on the course of gonorrheal arthritis.

2. Diligent search should be made for the point of focal infection, whether in the urinary tract or elsewhere.

3. Treatment must include thorough eradication of the focal infection if the arthritis is to be benefited.

4. The best results are obtained by antigonococcic serum, associated with the early use of Bier's hyperemia treatment.

5. Early rest and immobilization of the joint may be necessary.

In a limited number of cases surgical intervention is necessary, and, if indicated, should not be too long delayed.

CLINIC OF DR. SIDNEY STRAUSS

RUSH MEDICAL COLLEGE

CARDIAC ARHYTHMIA

Newer Studies on the Conduction System of the Heart. Analysis of the Venous Pulse by Means of the Polygraph. Analysis and Interpretation of the Different Cardiac Irregularities, with Special Reference to Clinical Manifestations, Prognosis, and Therapy.

THE subject which we take up today is a distinct branch of cardiac pathology, the study of which has only recently been begun. For many years we have studied the separate murmurs, analyzing and classifying them. During all these years we have simply noted that there was an arhythmia and let it go at that. Only when Mackenzie began his studies with his polygraph did we begin to realize that the separation of the different arhythmias might be as helpful in analyzing cardiac disease as the separation of the different murmurs had been with the advent of auscultation. It has taken us almost twenty years to realize that such analysis is worth while. The study of the arhythmias is simple, provided that we start from the beginning—that is, start with an understanding of the conduction system of the heart and of the waves of the venous pulse.

Conduction System.—Studies of His, Tawara, Keith and Flack, and Wenckebach have differentiated from the heart muscle certain muscle strands and bundles, the remains of the primitive cardiac tube, of a special type, over which the heart-beat is conducted. These muscle bundles are intimately mixed up with nerve bundles, so that these discoveries have not as yet solved the problem of the myogenic or neurogenic origin of the heart-beat. These muscles are, however, distinctive; can

be differentiated easily from the ordinary heart muscle, and form a definite system which I have tried to indicate *very diagrammatically* in Fig. 192.

It has been definitely shown that the heart-beat starts in the special bundle or node just at the junction of the superior vena cava with the auricle, described by Keith and Flack, and called the node of Keith and Flack, or the *sino-auricular node.* This region is called the sinus region, that is, the region between

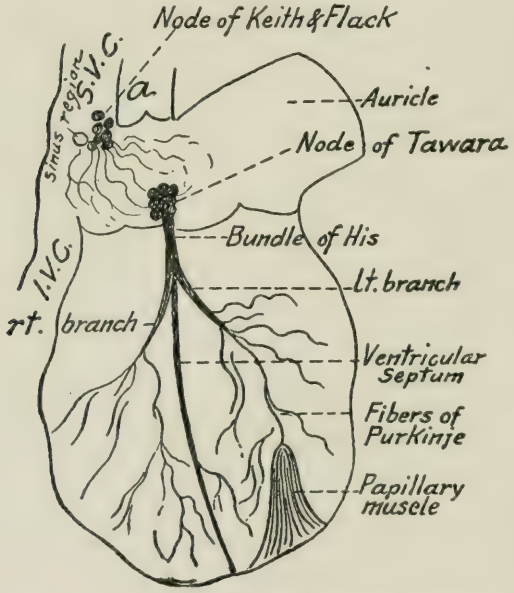


Fig. 192.—Conduction system, very diagrammatic.

the inferior and superior vena cava which in the embryo and in some of the lower animals forms a separate chamber of the heart. This sino-auricular node is also called the "pace maker," as in the normal rhythm this specialized bundle of muscle and nerves sets the pace for the heart.

The connection between the node of Keith and Flack and the second node is the only part of the conduction system which has not been worked out. There is still some dispute as to whether the impulse must traverse the whole auricle to reach

the node of Tawara, or whether it makes a short cut. At any rate, the impulse reaches the second node described by Tawara, and called the *aurivuloventricular node*. It is situated in the right auricle near the mouth of the coronary sinus. From this extends the *bundle of His*, or auriculoventricular bundle, which crosses the auriculoventricular septum, buries itself in the interventricular septum, divides here into two bundles, one to the left ventricle and one to the right, and then branching continuously threads its way throughout the whole cardiac musculature, including the papillary muscles, and ends here in the fine fibers of Purkinje. It is over this path that the cardiac impulse travels, and as this is the remains of the primitive cardiac tissue, the impulse may and often does arise in any part of this system, as we shall see later in taking up the different irregularities.

Venous Pulse.—Besides the knowledge of the conduction system it is necessary to understand the meaning of the venous pulse, in the light of the newer anlysis made by Mackenzie and others. First, it is necessary to forget the old teachings about positive and negative venous pulses and about one type coming from below and the other from above. By venous pulse we mean that pulsation which is often (in about 80 per cent. of all people) seen or recorded by instruments in the neck just above the sternoclavicular junction, in the jugular vein—as a rule, in the right jugular. This pulsation is made up of rises and falls which correspond to the rises and falls in pressure in the right auricle. In other words, by studying the jugular pulse we get a good idea as to what is going on in the auricle, just as by studying the arterial pulse we get some notion of ventricular action. Thus, you see, every venous pulsation, no matter of what type, is transmitted from the auricle and must come from below. We also have here an almost direct means of studying what is going on in the auricle, and in this lies the great value of analyzing the venous pulse. In order to analyze the venous pulse it must be compared with some other pulsations, the rises and falls of which can be definitely fixed, either the radial, the carotid, or the apex-beat. Hence, in studying the venous

pulse we need the polygraph, an instrument which will allow the recording of two or more pulses at the same time, so that one may be compared with the other.

As we stated before, the venous pulse is made up of various rises and falls or waves which bear definite relation to the rises and falls in the right auricle. We have first the *a* wave (Fig. 193), or auricular wave, due to the rise in pressure caused by the auricular systole. As the auricular systole immediately precedes by one-fifth of a second the ventricular systole, this wave in our comparison with the arterial pulse must be the one which just precedes the rise of the carotid or radial artery. The *a* wave is the wave which gives the venous pulse its normal

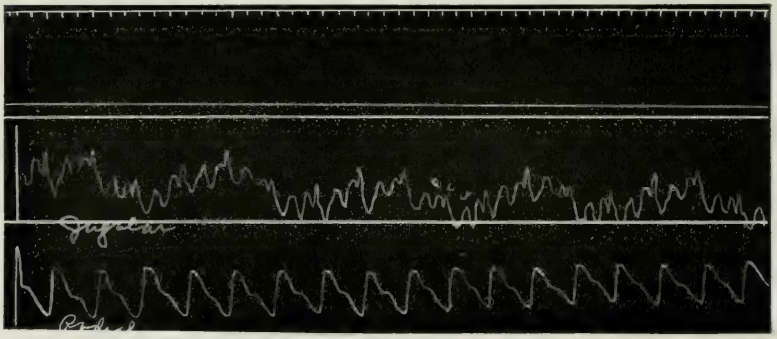


Fig. 193.—Normal venous pulse showing three normal waves (*a*, *c*, *v*).

tone, and the normal venous tracing is often called the auricular type of venous pulse, as opposed to the ventricular, which is abnormal and is characterized by the absence of the *a* wave.

Following the *a* wave there is a slight depression, followed in turn by a rise, the so-called *c*, or carotid wave. This wave was thought by Mackenzie to be transmitted to the jugular by the carotid; but the consensus of opinion and the evidence are all in favor of this wave being due to a rise in pressure in the auricle, caused by the closure of the tricuspid valve and the emptying of the coronary veins into the auricle. Next in order we have a deep depression, the *x* depression, followed by the third or *v* wave, the ventricular wave of Mackenzie, or ventricular stagnation wave of Hering. This wave begins about

the time of the closure of the semilunar valves and is related, although not definitely, to the dicrotic notch of the arterial pulse. It is supposed to be due to the stagnation in the ventricle which occurs at this time. Following this we have the second deep depression, *y*, due to the emptying of the auricle and lasting, as a rule, until the next auricular contraction. At times, however, in very slow pulses there is another rise described by Hirschfelder and Gibson, the *h* wave, supposed to be due to the flapping of the tricuspid valve in middiastole and corresponding to the third heart sound of Thayer (Fig. 194).

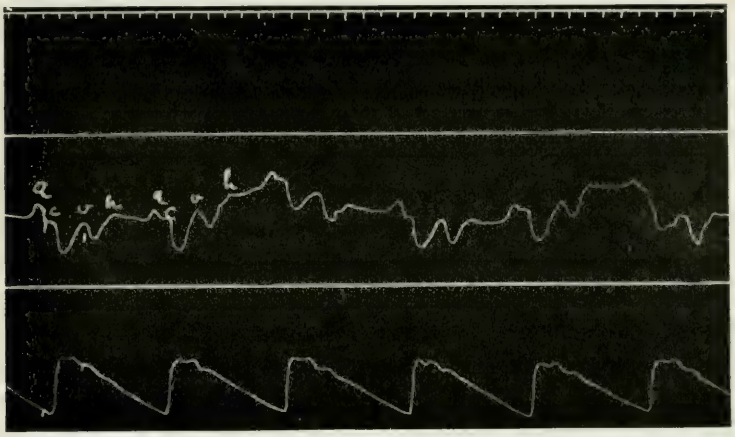


Fig. 194.—Normal venous pulse showing extra wave ("*h*").

Arhythmias.—Bearing these facts in mind, we may now proceed to an analysis of the arhythmias, and we shall find it a rather simple matter and not the bugbear which it may have seemed to one giving only a cursory glance at the subject. There are very few irregularities, and the following include practically all which you will meet:

Sinus arhythmias.
Extrasystoles (pulsus bigeminus).
Auricular fibrillation.
(Auricular flutter.)
Pulsus alternans.
Heart-block.
Paroxysmal tachycardia.

There may be a few arhythmias which cannot be placed under one of these heads, but they are very few, and thus you see that this phase of cardiac pathology, if studied properly, is no more complicated than the study of the murmurs, and may be just as advantageous in the diagnosis, treatment, and prognosis of the case.

Sinus arhythmia is the simplest and least harmful of all the irregularities. It is simply an irregularity in time, being made up of a series of fast and of slow beats. Other names for this arhythmia are "juvenile" and "respiratory," both being descriptive of certain forms that this irregularity takes. As its name implies, this arhythmia has to do with some change in the sinus region, which, as you know from our previous description, is that part of the heart between the venæ cavæ, but more particularly in the human heart that part which contains the node of Keith and Flack. This change is undoubtedly largely nervous in origin, due often to irritability of the vagus nerve. The irregularity varies often with respiration and occurs most frequently in young individuals with unstable nervous systems. It is a frequent occurrence in young people following a fever. It may also occur in older individuals who have an irritable nervous system, especially in the so-called vagotonic type of neurasthenic. There are, as a rule, no symptoms from this irregularity, and the prognosis both as to life and health is not affected in any way by its presence. It is well, however, to know that such an irregularity exists and that it occurs frequently in children and in young adults, so that when it does occur we may not be frightened by its appearance, but may reassure rather than frighten our patients.

Extrasystoles.—The second form of irregularity, extrasystoles, may or may not mean some grave cardiac trouble. First let us understand what we mean by the term "extrasystole." Extrasystole does not necessarily mean that we have more heart-beats than the normal, but that we have a beat whose origin is somewhere in the primitive cardiac tissue outside the pace-maker. In other words, an extrasystole may arise in any part of the conduction system (see Fig. 192) below the node of

Keith and Flack, the sino-auricular node. That is, it may arise in the auricle (Fig. 195), in the node of Tawara, in which case we have the true nodal rhythm, or in the ventricle (Fig. 196).

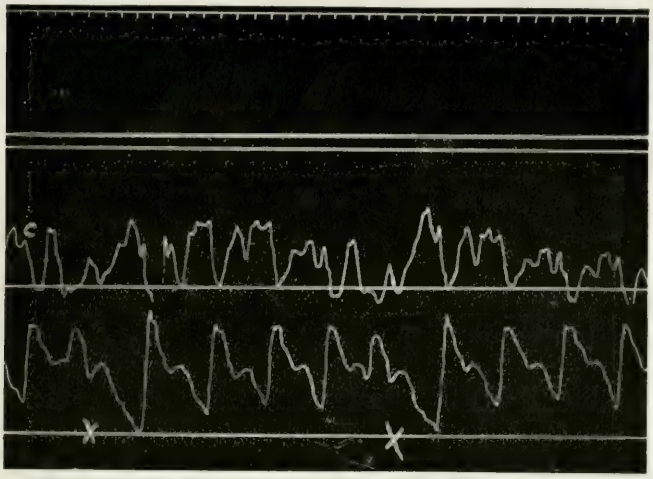


Fig. 195.—Auricular extrasystoles, showing two abnormal beats.

Another name for extrasystoles is *premature contraction*, given because the extrasystole usually follows the preceding

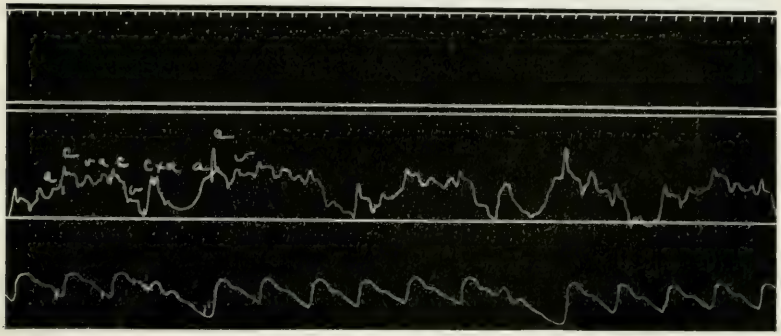


Fig 196.—Ventricular extrasystoles.

beat more quickly than normal. As with every normal or abnormal beat the heart makes there is a refractory period, when the time comes for the beat which should, in the normal,

succeed the extrasystole, we find the heart unresponsive to stimuli and this normal beat is dropped. There is, then, no beat until the time for the second normal beat after the premature contraction. Thus there must be a long pause after the extrasystole, and, as a matter of fact, the time taken by the normal beat and the extrasystole should equal the time taken by two normal beats, including the diastoles. This is a very good way of differentiating extrasystoles from other irregularities, and is very easily verified by using a compass on our tracings (Figs. 193–201). At times, however, the refractory period does not last until the time for the next normal period, and then

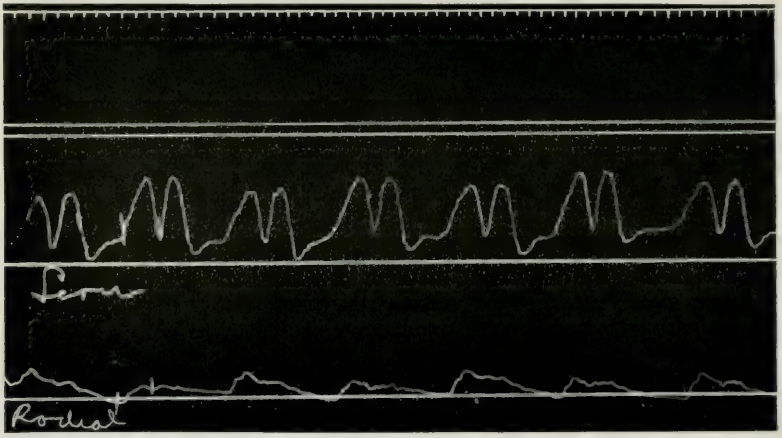


Fig. 197.—Pulsus bigeminus, showing distinct coupling in the liver pulse.

we have the normal beats going on as before, with an extrasystole inserted between two normal beats. This is called an interpolated extrasystole, and if there are many of them may confuse the diagnosis considerably.

The number of extrasystoles in different cases varies much. There may be only a rare one, which may be overlooked in a hurried examination, or they may occur regularly after every fifth, fourth, or even after every second beat, in which case we have the *pulsus bigeminus* (Fig. 197). The pulsus bigeminus, or coupled beats, occurs frequently following the administration of digitalis, and the exact nature of this pairing is still

somewhat in doubt. When it occurs after the use of digitalis it is a good sign either to stop or to lessen the dosage of that drug.

The question as to whether extrasystole is nervous or muscular in origin is still in dispute. The chances are that it may be either. We frequently have extrasystoles in arteriosclerotic cases, where they are undoubtedly due to the increased irritability of the muscular tissue around lesions in the myocardium. On the other hand, we frequently have extrasystoles or "dropped beats" or "intermittent pulse" in young people, or in smokers without any other sign of cardiac disease, and these may disappear when the patient becomes quieted or stops smoking. The best authorities seem to think that extrasystoles in themselves may or may not be of grave import. If they appear for the first time in patients over forty years of age they should not be passed over lightly. On the other hand, if they appear in young adults they may be of no consequence. In my opinion extrasystoles are always worth while taking notice of; it is true that many persons may have them for years and show no bad effects on the heart or on their general health, yet as we are doubtful of the pathology of this particular irregularity we are certainly doubtful as to just what it means. We can, however, reassure our patients, provided there are no other signs of cardiac disease, that this abnormality need cause them no worry, as it has been known to exist for years without causing any disturbance or hindering the activities of the bearer in any way. If there are other signs of cardiac or vascular disease, no matter how slight, we should be more guarded in our reassurance.

Extrasystoles are the one form of irregularity which may give symptoms of themselves. The patients often complain that the pulse is intermittent, or that they feel that the heart is dropping a beat, thus describing their sensations without feeling the pulse; or they may have a peculiar "fluttering" sensation or feeling of constriction in the precordium at the time of the extrasystoles, especially if these premature beats are at all numerous. These sensations may bring the patient to you, and

if the only finding is the extrasystole the only treatment he needs is your reassurance. There is no need for treatment of this irregularity *per se*.

Auricular Fibrillation.—The third type of arhythmia which we take up, auricular fibrillation, is of more serious import than the other two. It is probably the commonest of all the irregularities and has been studied more than any of them. It is called by the German school the *pulsus irregularis perpetuus*, and by the English the "absolutely irregular pulse," the latter term excellently describing the character of the pulse.

In this type of pulse we find no two beats alike (Fig. 198). There is absolute irregularity both as to time and force. On

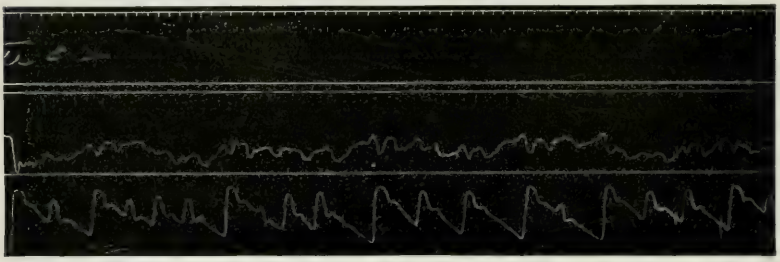


Fig. 198.—Auricular fibrillation, showing distinct ventricular venous pulse, and in the longer beats of the venous pulse the very small waves are seen superimposed upon the larger.

analyzing the tracings, we find the distance between no two beats the same, and we see the great variation in the height or force of the impulses in the radial pulse. Furthermore, on studying the jugular pulse we find an absence of the *a* wave with the *c* and *b* present, or with both *c* and *b* waves combined into one wave with a large plateau; in other words, a ventricular type of venous pulse, which is abnormal.

It was this absence of the *a* wave which led Mackenzie, before the advent of the electrocardiograph, to call this "nodal rhythm." His first thought was that, as there was no *a* wave, the auricle had become paralyzed; but as in a large number of cases in which this arhythmia occurs the auricle was hypertrophied, he discarded this theory. He then accounted for the

absence of the *a* wave by placing the origin of these beats in the node of Tawara, in which case the auricle and the ventricle would beat at the same time and only one wave would appear in the jugular tracing.

This theory was the prevailing one—even though Cushny had made the suggestion, based on findings in experimental animals, that this absolutely irregular pulse might be due to fibrillation of the auricles—until Lewis, in London, and Rothberger and Winterberg, in Vienna, showed conclusively with the electrocardiograph that it was indeed caused by just such fibrillations of the auricles as Cushny had seen in his animals. Since then this irregularity has been known as "auricular fibrillation." When we have such an irregularity, we have then no one single strong beat of the auricle, but the auricle is in a continuous worm-like motion, making numerous ineffectual small contractions. Fortunately, not all these contractions are strong enough to cause excitation of the node of Tawara or to be carried through to the ventricle, in which case we should have ventricular fibrillation and death. The beats are, however, in some way conducted through to the ventricle, but at very irregular intervals and with marked variation in force, giving us thus the absolutely irregular arterial pulse.

The *pathology* of this irregularity is not at all clear. It is undoubtedly due to some disturbance in the auricular tissue, but no distinctive lesion has been found. It occurs most commonly in mitral lesions and especially in mitral stenosis. It may, however, occur in myocarditis, from an old arteriosclerosis, or syphilis, and also follows at times acute febrile disease, especially pneumonia. It has been called the "perpetually irregular pulse" because when it once occurs it usually persists. It may, however, be very transient. I have seen it occur following a pneumonia, last only a few days and disappear, leaving apparently no bad effects.

As we said before, this irregularity is of serious import. It means some disturbance and usually a permanent disturbance of the auricle. Auricular fibrillation in itself does not mean immediate death—often far from it, as patients may live for

years with such an irregularity. Its appearance undoubtedly adds some gravity to the prognosis in an organic heart lesion, as a heart whose auricle is fibrillating is not working to as good advantage as a heart which is beating regularly, and the failure of compensation, other things being equal, is bound to come sooner. Auricular fibrillation does not enable us to be any more accurate in our prognosis as to the duration of life, and we should be careful in making absolutely bad prognoses, or in causing our patients to lead a too sedentary life just because of the appearance of the absolutely irregular pulse. The irregularity is only one of the signs on which we base our prognosis.

There are no special symptoms caused by auricular fibrillation. There is no special treatment for this irregularity. Although there seems to be abroad the idea that the appearance of this irregularity calls for the administration of digitalis or its derivatives, the fact still remains that the one and only indication for the exhibition of digitalis is a failure of compensation. Auricular fibrillation in itself is not a sign of failure of compensation. It is true that it usually accompanies or is accompanied by such failure, and in these cases digitalis is indicated. It is in these cases with auricular fibrillation that digitalis has its best effect, as Mackenzie has shown in his admirable treatise on digitalis; that does not signify that we should reserve the use of digitalis for auricular fibrillation, as one cardiologist has suggested, or that digitalis is equally effective in all auricular fibrillations, no matter what the etiology. Digitalis often does little good in failure of compensation secondary to an exophthalmic goiter or to an arteriosclerotic myocarditis, even though accompanied by an auricular fibrillation. Let us not forget that it is the failure of compensation which is helped by the digitalis, but that this failure, when accompanied by auricular fibrillation, often responds much more readily and more brilliantly to the drug than in cases without this irregularity, provided, again, that the fibrillation appears in a case of rheumatic mitral stenosis.

Auricular Flutter.—Closely related to auricular fibrillation

is auricular flutter. It is, as a rule, very easily differentiated. Very often in auricular flutter there is no actual irregularity of the arterial pulse, or if there is an irregularity it is one only of time and not of force and the time intervals bear definite relations to one another. The reason for this is seen readily if we consider the cause of the irregularity. Here again we have the absence of one single auricular wave for each ventricular complex. Instead we have an extreme tachycardia of the auricle up to 200 or 300 a minute. The contractions of the auricle are not as large or as strong as normal, but still not nearly so slight or ineffectual as in fibrillation. Some of these contractions of the auricle are blocked or are not strong enough to go through, and we have the ventricle beating only with every second, third, fourth, etc., beat of the auricle. At one time, within short intervals, it may follow the second, at an-

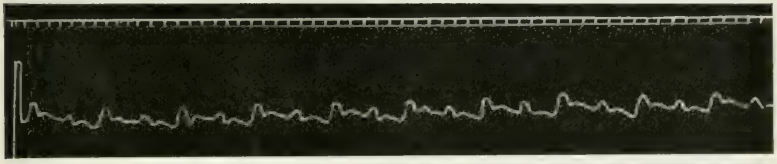


Fig. 199.—Pulsus alternans.

other the third, and thus give us the irregularity in time mentioned above. Often this irregularity is accompanied by a tachycardia of the ventricle, but rarely is the ventricular rate equal to the auricular. Digitalis very often is effective in stopping this irregularity—often converting it first into an auricular fibrillation, which in turn becomes normal.

Pulsus Alternans.—The alternating pulse, "pulsus alternans," as distinguished from the foregoing, is an irregularity of force and not of time (Fig. 199). Every other beat is a weak one. The interval between each beat is always the same, but you will notice in the radial pulse that the height or force of every other beat is less than that which goes before it. This is to be distinguished from the "pulsus bigeminus," in which every other beat may be small, but in which there is also an irregularity in time (Fig. 197). The cause of the pulsus alternans is not known, but

it has been assumed that it is due to some disturbance in the myocardium. The prognosis is grave. If the irregularity continues for any length of time the patient usually succumbs. At times the patient may recover, but this irregularity is always of grave significance. If there is failure of compensation, digitalis may help, and the irregularity may pass away with the return of compensation; as a rule, however, when this irregularity appears without any apparent cause, compensation is not restored.

Heart-block.—The last two types which we consider are really not arhythmias at all in the strict sense of the word. In heart-block the radial pulse is usually regular, but very slow

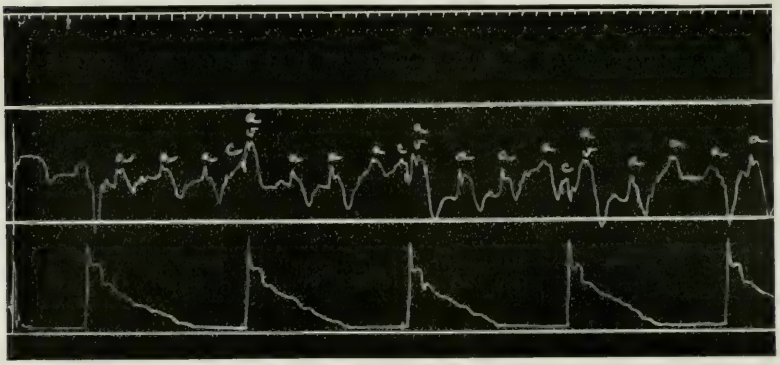


Fig. 200.—Heart-block, four auricular beats to one ventricular.

(Fig. 200). On the other hand, we find the auricle beating at the usual rate. There is some lesion in the bundle of His (Fig. 192) which blocks the conduction of the beat from the auricle to the ventricle. This block may be complete, in which case the auricles and ventricles beat independently—that is, the real arterial pulse or ventricular beat arises in the ventricle itself, in some part of the primitive cardiac tube. The slowness of the beat, usually 32 or less, in complete heart-block is accounted for by the fact that the farther away from the sinus region the beat arises, the slower is the basic rhythm.

The lesion or disturbance in the bundle of His may, however, not be so great as to block all the beats, and we may have

every other beat or every third or fourth beat going through. In this case we say we have a partial heart-block. The beats of the ventricle arise, then, in the normal way, are stimulated through the normal conducting system, but the ventricle responds only to every second, third, or fourth beat of the auricle, as the case may be. The simplest form of block, or the beginning of heart-block, consists of a delay in the impulse through the bundle. In that case we have a prolonged *a–c* interval; that is, the time between the auricular and ventricular systole is prolonged, and this time is taken up almost entirely by the traversal of the bundle and its branches. The *a–c*

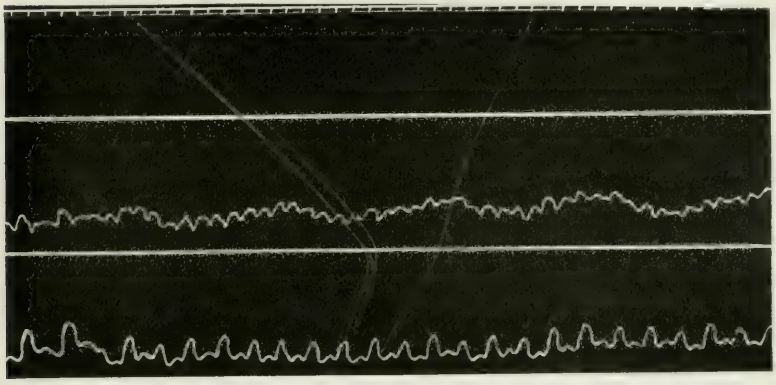


Fig. 201.—Paroxysmal tachycardia, rate 150.

interval is normally one-fifth of a second. In these cases of earliest block it may be doubled or trebled.

In heart-block, as we have said, there is usually some organic lesion of the bundles; rarely no lesion has been demonstrated. Digitalis may cause a temporary block and often causes a prolonged *a–c* interval, due most likely to its action on the vagus. The treatment of heart-block *per se* is of no avail. If due to syphilis, antisyphilitic treatment may clear up the trouble or at least ameliorate it. In partial heart-block digitalis should be used very cautiously, as it may turn a partial into a complete heart-block and bring on an attack of Stokes-Adams syndrome or a sudden stoppage of the ventricle, which may fail to take up

its own rhythm. The prognosis of complete heart-block is bad, although patients may live for a number of years after the onset.

Paroxysmal Tachycardia.—The last abnormality which we shall consider, "paroxysmal tachycardia" (Fig. 201), is very rare and very little of a definite nature is known about it. As the name indicates, it is characterized by a very rapid pulse, varying from 140 to 300, coming on in paroxysms with an acute onset and abrupt ending. The paroxysms vary in length from a few minutes to a few weeks or, rarely, a month or more. This phenomenon may occur in an otherwise perfectly normal heart or may be associated with an organic valvular or myocardial lesion. In the latter case one must be careful not to confuse it with a simple tachycardia whose incidence and subsidence is usually gradual.

The *cause* of these attacks is still in dispute, some leaning to the myogenic and others to the neurogenic origin. In cases which have come to autopsy both extensive lesions of the myocardium and lesions of the vagus and the cardiac center in the brain have been found. It seems likely that the cardiac nerves play a large part, as these attacks are occasionally aborted and very often stopped by certain procedures, such as deep breathing, swallowing large capsules or cachets, pressure on the vagi, vomiting and belching, which act reflexly on the vagus. On the other hand, in most cases of paroxysmal tachycardia the beats arise in some part of the heart other than the pace-maker, and consist of a series of extrasystoles coming on in rapid succession. In other words, some part of the heart, either the auricle, the ventricle, or the auriculoventricular node, has become highly irritable and responds to stimuli very much more rapidly than in the normal. In a given individual all paroxysms are alike, as a rule indicating that they must all start in the same focus. This would lead us to believe that there was some lesion at this focus giving rise to this hyperirritability. This lesion, however, does not explain the paroxysmal character of the attacks.

The *symptoms* of such attacks are very few, although every patient can tell you when his attack begins and when it ends.

At times there are a few premonitory signs due probably to the occurrence of a few preliminary extrasystoles before the attack begins. Usually the patient complains of palpitation and an uneasy feeling in the precordium. If the attack lasts only a few seconds or minutes it leaves the patient none the worse for it. The longer paroxysms usually cause marked weakness, and may also cause the patient precordial pains, with the sense of constriction and pressure in the chest. In most cases these attacks pass off without any permanent damage. In some cases, however, the heart muscle becomes insufficient and patients have died during the attacks. In those cases in which there is no demonstrable organic cardiac lesion there are no signs in the heart—most remarkable, there is, as a rule, no cardiac dilatation.

Treatment, as a rule, is of no avail. Various drugs have been tried without any effect. Various methods of stimulating the vagi, as described above, have also been tried, and when the time comes for the paroxysm to end may assist in ending it. Some patients can stop their attacks by taking deep breaths, others by vomiting or by swallowing large objects, but these procedures may be tried many times during the paroxysms without avail until it is finally time for the attack to stop of itself. The patient should be kept at rest during the attack with an ice-bag over the heart. If there are signs of failure of compensation digitalis should be given, otherwise the best method is to let the patient alone. The prognosis is, as a rule, good.

The paroxysms usually end as they begin. In the prolonged attacks there is, of course, the danger of collapse of the myocardium; we are helpless to prevent this. All one can do is to assure such patients that these attacks usually pass over, and after a few attacks the patient learns this for himself.

Conclusion.—This ends our discussion of the irregularities. We have seen that there are comparatively few types; that these are well defined and with a little study can be differentiated; that when differentiated they help us in making the diagnosis and prognosis and in giving the proper treatment.

We must remember, however, that the arhythmia is simply one of the signs of cardiac disease, and that, in order to arrive at the proper conclusions, we must consider all the signs and symptoms which make the complete picture in any given case. Remembering this, the study and analysis of any irregularity which we meet will be of great help to us in coming to the correct conclusion.

DR. RALPH C. HAMILL,
30 N MICHIGAN BOULEVARD,
CHICAGO.

April 24, 1917.

W. B. Saunders Company,
West Washington Square,
Philadelphia, Pa.

Dear Sirs:-

I have your favor regarding my clinic entitled "Some Considerations of the Problems of Psychiatry", which appeared in the January 1917 number of the "Medical Clinics of Chicago."

I note with regret that some of my medical colleagues take exception on religious grounds to certain statements I make in the clinic in question.

I am distressed exceedingly at the carelessness on my part in the choice of words. I had not the slightest intention to offend in any way, to speak irreverently of any faith, or cast any aspersions on any religious belief. My sole aim was to show the peculiar workings of the unbalanced mind and the association of ideas when influenced by the religious impulse..

I trust my colleagues in the profession, and yourselves will accept this explanation in the good faith in which it is given.

Very truly yours,

Ralph C. Hamill

NOTE. The above letter from Dr. Ralph C. Hamill speaks for itself. Of course it is understood that the Publishers likewise regret the above incident, as they would in no way or at any time cast any aspersions upon the religious faith of any one.

CLINIC OF DR. RALPH C. HAMILL

NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

TABES DORSALIS

Symptomatology in Detail. Differential Diagnosis and Treatment.

December 18, 1916.

THERE are a few nervous diseases that any man ought to be able to recognize by just looking at them. Paralysis agitans, which I demonstrated before, is such a disease. Another very common disease is locomotor ataxia, or tabes dorsalis. Medical men see many more cases of tabes than of paralysis agitans.

This patient today is of particular interest in the matter of "snap-shot diagnosis" merely by the characteristics of his gait and appearance. The only condition which one meets at all frequently with a gait similar to that of locomotor ataxia is the gait seen in combined degeneration of the cord associated with pernicious anemia.

DR. HAMILL: What characteristic things do you see in this man's gait as he walks across the room?

VISITING PHYSICIAN: He throws his feet forward against the floor; he lifts his legs high and comes down on his heels, and when he closes his eyes he is very unsteady. His feet are far apart and there is hyperextension of the legs.

DR. HAMILL: Is there anything else you can tell about his gait?

VISITING PHYSICIAN: He is uncertain about his step and walks sideways.

DR. HAMILL: That covers the most important elements of the gait. Perhaps the first thing to be said about it is that the muscles are all used, but not to their best ability. There is

no paralysis anywhere, but there is not good control over the relationship of the muscles that oppose one another. As he throws his foot forward he throws it too far and then he stamps it down. It has been said that it is characterized by the fact that he puts his heel down first, but we all do that. He jabs his heel down on the floor, and at the time he hits the floor with his heel he is pulling the foot back because he has thrown it too far forward. Another characteristic is that it might be called a tractor gait rather than a propellor gait. We ordinarily walk with a little push from the calf muscles; we don't have to fall from one foot to the other, but we carry our body steadily forward. The reason for this abnormality lies in the fact that the tabetic does not get the information from his legs that the normal person does, with the consequence that he, in the first place, throws his feet too far; in the second place he does not get off of his balance before things are all ready for him. He must have something there to settle on before he can leave the leg that he is standing on, so his walking apparatus is always in front of him. In a very high degree of ataxia these patients walk with their hips away back and their heads well over their feet, so that they can see them all the time. In a highly typical gait, where the patients have to use a cane, you see this movement (demonstrating).

It has been mentioned that this man's gait is very much worse when his eyes are closed. Now that gait is something that you should be able to recognize wherever you see it. It should immediately stamp the man or woman as a sufferer from disease of the posterior columns, and disease of the posterior columns in nine cases out of ten means tabes. The tenth case is usually combined degeneration of pernicious anemia. This man can walk on the ball of his foot, but it requires very much greater effort and is marked by an increasing clumsiness.

The anterior horns are the central origin of the fibers that go directly to the muscle. These impulses are all going in a downward direction and are of the nature of the motor impulses. The anterior horns are innervated by the pyramidal fibers that come from the motor cortex. They run to that level in the

cord at which the anterior horn cells lie for which they are destined and then leave the lateral tract to arborize; down the pyramidal tract are coming voluntary motor impulses of probably a rather high degree of co-ordination. The motor co-ordination has all been established before the anterior horn cells are reached, and the proper impulses are given to the cells in the anterior horn so as to bring about a voluntary act. There is a definite time taken for that impulse to leave the cortex and pass down the pyramidal tract all the way to the muscle. Then in that muscle is set up the mechanism which causes that muscle to contract. The posterior column has fibers coming to it from that same muscle. These fibers come in in some such manner as this (illustrating on blackboard). They are the carriers of sensory impulses arising from the skin, muscles, and the joints. The impulses pass into the posterior ganglion and undergo some change there in all probability, and then pass on into the posterior horn, undergoing as likely as not some change in the gray matter at the entrance to the posterior horn, then on into the posterior columns. Thus these impulses are all passing away from the muscle toward the central nervous system instead of toward the muscle. Hence, there is no reason to suspect paralysis from a posterior root involvement, whereas there is every reason to suspect paralysis from an anterior involvement.

To go a little more into this patient's history. He is now fifty-four years of age. He had a chancre about twenty-five or thirty years ago. He was apparently well until eighteen years ago, when he commenced to have a pain in the abdomen, with a desire to evacuate the bowels after eating, but did not always get a stool. About the same time he noticed what are described as "lightning pains" in the legs. These might come on at any time, driving him to bed, or they might come on while he was asleep and awaken him. Any symptom of syphilis of the central nervous system may be found in locomotor ataxia, although not necessarily a characteristic of locomotor ataxia. Early symptoms are frequently diplopia, severe headache, difficulty with vision due to a dilated pupil, with paralysis of the iris, retention of

urine—any one of these symptoms may be found in the early history of locomotor ataxia, and merely speaks of the involvement of the central nervous system by syphilis.

About fourteen years ago this patient noticed that it was difficult for him to walk in the dark or to follow a straight line, and this condition has gradually increased. It is now very difficult for him to hold his equilibrium with his eyes closed and sufficiently difficult with them open.

Nine years ago a ptosis of the right eyelid developed over night. This cleared up after taking potassium iodid and mercury for three months. Ptosis is often a sign of very early involvement of the central nervous system. At about the same time he noticed a difficulty in swallowing. He would have a strangling sensation in his throat which would waken him, and it was sometimes difficult for him to breathe. The thing that brought him to the hospital was that two weeks ago he had what he described as a "strained" tongue; he had difficulty in articulating. He knew what he wanted to say, but had to talk slowly. A swelling appeared on each side of his throat which lasted two or three days.

Examination reveals that the pupillary reflexes to light are gone. He has what are termed "Argyll Robertson pupils." They are not the typical Argyll Robertson pupils. He has a loss of knee- and ankle-jerks, a normal plantar reflex—which is of importance in differentiating this condition from combined degeneration, in which there is also an involvement of the pyramidal tract with a Babinski sign. He also has various sensory disturbances. The most marked and readily demonstrable is a diminution amounting to an almost complete loss of joint and tendon sense in his toes and feet. There are also disturbances of touch and pain, especially deep pain. I can take his Achilles' tendon between my thumb and middle finger and squeeze it as hard as I can without any complaint of pain from him.

He has a peculiar symptom, namely, the strangling in his sleep, which came on about eight years ago. This strangling is not present now, but what is present as a remainder from the lesion is a very peculiar voice, which suggests the voice at the

age of puberty. It is very apt to break if he gets excited and talks rapidly. On examination with the laryngoscope, there is no paralysis of the vocal cords, but an ataxia thereof. They will not obey a voluntary impulse in a co-ordinate manner. This is an unusual symptom of locomotor ataxia; I have only seen it in one other patient.

Etiology and Pathology of Tabes.—The etiology of locomotor ataxia is unquestionably syphilis. Up to the time that the Wassermann reaction was perfected, until we were able to find the spirochete in the spinal cord, there was some question as to whether it was due to syphilis. Syphilis affects the spinal cord in a peculiar way when locomotor ataxia is the outcome. In practically every case of locomotor ataxia if a careful search is made a slight meningitis around the posterior columns is detectable. The soft meninges that surround the spinal cord and convey the smaller blood-vessels will be found to be somewhat thickened across the posterior columns. They may be somewhat thickened around the anterior columns. That thickening may be accompanied by a slight round-celled infiltration, especially around the vessels. The vessel walls themselves are thickened. Instead of a vessel being in its normal contour and thickness the intima will be seen to be irregular in outline and decidedly thickened. The elastic membrane, instead of being just a single layer, will perhaps show a breaking and the adventitia will be thickened. The thickening may be uniform all the way around or it may be more in one place than in another. The small round cells sometimes found show a chronic low-grade inflammation. As to just how that inflammation affects the parenchyma of the central nervous system so as to produce the lesions found in tabes is in dispute. As far back as 1892 Nageotte claimed that this was due to a pinching of the posterior root fibers as they enter the cord, pinching of the chronically inflamed soft meninges at that point.

The nerve-fibers which come in from the ganglion bipolar cells are the central fibers. Some of these fibers upon entering the spinal cord run up in the posterior column. As you know, according to the law of Waller, degeneration takes place when

the root fiber is cut off from its cell of origin. Consequently, when a pinching by the inflamed meninges takes place at the point of entrance of the root fiber into the spinal cord, degeneration is set up in that fiber and we get the posterior column change of locomotor ataxia.

January 15, 1917.

At the last clinic you will remember that I began a discussion of locomotor ataxia. I am spending a good deal of time on this subject because syphilis of the central nervous system, including locomotor ataxia, forms a considerable part of the nervous diseases that a physician is called upon to diagnose and treat. I hope that by getting before you a clear idea of the genesis of the disease you will be enabled to think about the disease in other terms than mere parrot-like memory.

I think the most reasonable way to think of the symptoms of locomotor ataxia is on the basis of two facts: First, there is a chronic meningitis on the posterior surface of the cord. As to just why this meningitis is more inclined to demonstrate itself on the posterior surface rather than on the anterior surface or all the way around I cannot say, but that is a fact which you have to remember. Second, when a nerve-fiber is cut off from its cell of origin it undergoes degeneration away from that cell. If you remember these two facts you have a fair idea of the pathologic picture of locomotor ataxia. The posterior meningitis affects the root fibers either through a toxic action or through a mechanical action. All scar-tissue contracts, hence the diseased meninges would contract about the surrounded roots—the root-fibers which come from the bipolar cells in the posterior ganglion and run through the posterior root. The loss of reflexes and the pathologic picture which always shows the degeneration of the posterior column indicates that this fiber has been cut.

One of the first changes in locomotor ataxia is a loss of joint and tendon sense. Now joint and tendon sense means sense of position. Sense of position requires information from the structures around the joint. For instance, if the joint is straight the skin is in a certain position; if the joint is crooked the skin is in another position; if it

is straight the tendons are in a certain position; if it is crooked they are in a different position; if the joint is straight the bones are in a certain position; if it is crooked they are in a different position—in other words, if we say that joint and tendon sense is dependent upon all these factors, that is practically the same as to say that it is necessary to have information about all these structures in order to know their position. It is quite reasonable to suppose that if something mechanical is affecting the posterior roots it is going first of all to diminish that function which is most complicated. It is going to first of all produce a generalized diminished function from that posterior root, so that the information *in toto* will not be as good as it should be in order to give the proper news to the complicated organism in the central nervous system. If we think of locomotor ataxia in these terms it seems to me that we can reason out a little bit why the symptomatology is as it is. It is not at all difficult to think that a mere touch on the skin is a very much more simple thing to report to the central nervous system than the position of a joint and its movements.

It has been claimed that one of the very earliest symptoms of locomotor ataxia is the loss of vibratory sense. Vibratory sense is, of course, a very complicated sense. It depends upon very slight differences in sensation being reported to the central nervous system with tremendous frequency.

Locomotor ataxia is a system disease. Multiple sclerosis is not a system disease; there are certain points of sclerosis at different points, and it is rather haphazard in its symptomatology. Locomotor ataxia, on the other hand, has a pretty definite origin and leads to definite results that give you some information as to the physiology and the normal function of the central nervous system.

There is one other thing about the inflammatory stage of locomotor ataxia in the early stages that we have to bear in mind. Inflammation in any part of the body is practically always associated with one phenomenon, and that is pain. So in locomotor ataxia the most general early symptom is pain. Just why that pain comes in crises, comes momentarily, and

then disappears, with intervals of perfect freedom in between, we do not know. That it does not rule out a chronic condition is not difficult to understand when you think that epilepsy or migraine are manifestations of some constitutional disease, so the nature of the pain in locomotor ataxia, its coming and going, its momentary duration, does not rule out the idea that it is due to a chronic, constant inflammatory process. The pains of locomotor ataxia are such that they frequently are not reported by the patient. There may be a few jabs quickly repeated, then freedom, a few more, and an interval of a week or month before the appearance of more jabs. On the other hand, they may have a few jabs every day, but the pains themselves are not incapacitating, and so frequently there is no complaint of pain. The patient I expect to show you today did not complain of pain until she was asked about it, and yet she had had the typical jabs of pain. It sometimes happens that in locomotor ataxia there is more or less constant pain. It may be in the nature of backache—it may be described as lumbago.

This patient, a woman fifty-four years of age, has been married ten years and is a laundress by occupation. This patient's family history is uneventful. She knows of no family infection. She has had one child and no miscarriages. She came to the hospital complaining of loss of eyesight, which she first noticed three months ago or, rather, it was called to her attention at that time when she was told that she was not getting the clothes as clean as she previously did. Upon inquiry it developed that she had had "rheumatic" pains in the legs and shoulders for about ten years. These would come about once a month and were accompanied by no fever and no sore throat.

DR. HAMILL (to Patient): What sort of pains were they?

PATIENT: Just sticking pains like a needle and just as sharp.

DR. HAMILL: How long would they last?

PATIENT: For a couple of minutes and then another would come.

DR. HAMILL: When would they come?

PATIENT: Every time I caught cold, but not in the summer.

DR. HAMILL: Where would they come?

PATIENT: Down in the fat part of my leg.

DR. HAMILL: Would the leg be sore after the pain?

PATIENT: Yes, I couldn't touch it.

DR. HAMILL: Would the spot be sore when the pains would begin?

PATIENT: No, but I could not touch it afterward. They were just like lightning.

DR. HAMILL: Did you have any trouble controlling your water?

PATIENT: A little bit about holding it sometimes when I caught cold.

DR. HAMILL: How long have you had that trouble?

PATIENT: Only a year.

DR. HAMILL: Do you sometimes wet your clothes?

PATIENT: Yes, sometimes if I have a bad cold.

DR. HAMILL: The principal thing that is to be noticed about her walking is that as she turned around there was a little swaying just as she started back. She was not getting sufficient information from her joints and tendons to enable her to execute that movement. As she went up stairs she lifted her foot at least 6 inches higher than she needed to before she set it down. Then as she came down she set her foot securely on the lower step before leaving the upper step. That brings out a characteristic point in connection with locomotor ataxia; it is more difficult for these patients to go downstairs than upstairs. That is because in going upstairs the next step is nearer to the point of vision than the other step, but on going downstairs the next step is just that much further away. Another side of it is that in locomotor ataxia one of the most difficult things that the patients have to do is to relax their muscles—to allow the antagonistic actions just the right amount of strength. In going downstairs we have to relax the leg that stays behind just the right amount, and just as soon as the foot disappears from view they are not informed about it, and so they do not know just how far to drop themselves for the next step.

Laboratory Findings.—The Wassermann reaction was positive on both blood and spinal fluid; the Nonne, Ross-Jones, and

Noguchi reactions were also positive. There were 30 cells per cubic millimeter in the spinal fluid.

She complains most of all of the failure of eyesight. The pupils do not react to light, but there is reaction to accommodation. There is a primary atrophy of the disks such as is characteristic of locomotor ataxia or late syphilis. Her plantar reflexes are normal; there is no knee-jerk, no Achilles' jerk. There is no wrist-jerk in the left arm and no triceps-jerk, and there is no wrist-jerk on the right side. As for sensory changes, the only thing I have been able to demonstrate is a loss of joint and tendon sense in the great toes.

She performs the simple tests pretty well, but you could all see that there was the euphoria—the failure to realize the seriousness of her condition. That is one of the early symptoms of paresis, which, as you know, is another unfortunate outcome of late syphilis.

The diagnosis in her case is not difficult; the primary optic atrophy is, in the majority of cases, due to late syphilis. Such pains as she is having are typical of late syphilis—the sticking, lightning pains. This woman has a typical optic atrophy, loss of joint and tendon sense, and difficulty in handling herself as she walks, especially going up and down stairs, which is further evidence of the failure of joint and tendon sense.

January 28, 1917.

Today I want to show you some cases of early tabes. This patient is a man aged forty-five years, single, who was infected with syphilis seven years ago. In March, 1916, he began having sharp pains in the left knee and foot and difficulty in holding his urine.

If you watch him carefully as he walks you will see that as he turns around there is a little swaying back and forth of his hips. The turning is not natural and easy as in the case of a normal person. (Has patient walk with his eyes shut, taking five steps.) You can notice how different this is from normality, and yet as he walks with his eyes open you have to watch very carefully to appreciate any deviation from the normal.

Upon examination we find, first of all, rather small pupils, both of which are slightly irregular in form. There is practically no reaction to light, but quite a good reaction to convergence. They are almost typical Argyll Robertson pupils. As he stands with his toes and heels together with his eyes open he stands fairly well, but the instant he shuts his eyes there is a positive Romberg sign. All the old literature is filled with reference to the knee-jerk in locomotor ataxia. This is located in the first two or three lumbar segments. In tabes, if you remember, the vast majority start in with symptoms referable to the lowest end of the spinal cord. Bladder and rectal disturbance are very common early symptoms. Sensations around the genitalia, numbness, and that sort of thing are very common among the early symptoms. Those parts of the body supplied by the lowest end of the spinal cord are the parts most affected. Consequently it is reasonable to suppose that there will be disturbance of the ankle-jerk before we get disturbance of the knee-jerk, the ankle-jerk being supplied by the first two or three sacral, as against the first two or three lumbar, segments. There is no knee-jerk at all in this case, and absolutely no Achilles' jerk.

I can use every bit of strength in both my hands in squeezing his Achilles' tendon and he does not complain. Considerable pressure can be exerted on the testes without complaint of pain. The finger-to-nose test he performs fairly well, but there is a little uncertainty in the left hand. The wrist-jerks and biceps-jerks are present; the jaw-jerk is present.

There is one other thing which is one of the most characteristic things in this case (testing patient's chest with pin-point and match). There is some difference on his chest so far as pain sense is concerned; he feels the pin prick better on the upper part of his chest.

Case 2.—This man is forty-two, married at twenty-one, divorced seven years ago. One child died at seven months. He had gonorrhea at twenty years of age and has had it three times in the past fifteen years. He has had tonsillitis off and on for the last ten years, lasting only for a day or two. That is a little suspicious. Tonsillitis, as you know, lasts for a week or

two as a rule. A little soreness of the throat which only lasts for a day or two may very well be a specific throat. Six years ago he had "rheumatism"; that is, sharp jabs of pain, mostly in his ankles. He would have about one paroxysm a week, and these paroxysms have been more severe for the past two years. There has been some diminution of sexual power, and about a year ago he developed trophic ulcers in the soles of both feet. He has since had his great toes amputated. There is analgesia of the Achilles tendon and the testicles. There is a band of anesthesia around the chest. The blood Wassermann gave a strongly positive reaction, spinal fluid negative, 7 cells to the cubic millimeter.

In walking across the room, just as he turned and started back he did not take a normal step; there was just a little turning of the right foot as the movement got complex. As he shuts his eyes and turns to come back there is pronounced swaying. There is a slight reaction to light in the right pupil, none in the left. It is much better to accommodation. The right pupil is larger than the left. There is a very active knee-jerk on the right and also on the left, really a pathologic knee-jerk—it is *too* active. There is just a suggestion of an ankle-jerk in the left foot and none in the right. You see there is a very marked inconsistency between his knee-jerks and ankle-jerks. Knee-jerks are brisk, ankle-jerk barely discernible on the left, absent on the right. I can put all of my strength into my thumbs in squeezing his Achilles' tendon and he does not complain at all. There is distinct tendo achillis analgesia and also testicular. As I said, he has had both great toes removed, and he did not take any anesthetics for either operation and says he did not suffer any pain. Repeated jabs with a pin over his shinbone brings no complaint of pain, but at about the distribution of the second or third lumbar segment he appreciates the pinpoint. On his chest is a band of analgesia which is about 4 inches in width, starting at about the level of the nipples.

VISITOR: How about the marked knee-jerk and the anesthesia below?

DR. HAMILL: The most common lesion of syphilis is a

meningomyelitis. He can have a little sclerosis of the pyramidal tract or a little meningomyelitis causing pressure on the pyramidal tract, and then down below have a posterior meningitis sufficiently marked around the posterior roots of those fibers going to supply his ankle-jerk.

This man gets through the finger-to-nose test very well. The deep reflexes in the arms are very brisk, the jaw-jerk is present. He has a good deal of subjective numbness in the feet.

Case 3.—As this man walks across the room his gait is ataxic. He stands fairly well with his eyes open, but there is some increased swaying as he closes them. The right pupil is considerably larger than the left, neither reacts to light, but there is some reaction to convergence. There is a pretty fair knee-jerk on the right side. On the left side there is a good knee-jerk. There is no ankle-jerk whatsoever and I cannot hurt him in squeezing the tendo achillis.

He is forty years of age, married at twenty-five, has five children who are well. His wife had two miscarriages. He had a chancre at twenty-one; no secondaries. The chancre was burned off and he took medicine internally for about a month; he says both pills and drops—probably mercury and potassium iodid. Six weeks ago he had a sudden attack of severe pain down his right sciatic nerve. He has been perfectly well for nineteen years so far as active syphilis is concerned. Six weeks ago this sudden attack of pain set in; sharp jabs of pain, especially on movement. Four days ago pain appeared in the right shoulder, especially on movement. He was given an injection of cold salt solution into the sciatic nerve the night he entered the hospital. I saw him the next morning, and his right pupil was twice the size of his left, both were irregular in shape, and just for want of a little observation he had been sized up and treated as a sciatic. The tabetic side of the case had been entirely overlooked. The spinal fluid showed 30 cells to the cubic millimeter and the Wassermann was reported negative on both blood and spinal fluid on December 20th. On January 11th there were 8 cells to the cubic millimeter, blood Wassermann

triple plus (+ + +), spinal fluid positive. It is not reasonable to suppose that there would be that change in so short a space of time, and this bears out what I said before about the unreliability of the Wassermann tests.

Case 4.—This man walks fairly well with his eyes open. With his eyes closed there was just the one step which showed he started to lose his balance and he threw his feet out to regain his balance. With his heels and toes together he stands well, and there is very little increase in swaying as he closes his eyes. The pupils seem to be perfectly fixed to light, but they react to accommodation. The right reacts a little bit better than the left. There is a good knee-jerk on the right, a little increase in the one on the left. There is a good ankle-jerk on both sides. This is better, however, on the right than on the left, but there is very little difference. There is a very slight wrist-jerk on the right side, considerably better on the left. The biceps-jerk seems to be about normal, as is also the triceps. He has no tendon analgesia; on the other hand, he has quite marked testicular analgesia. He has 14 cells per cubic millimeter of spinal fluid. The Wassermann was declared negative on both blood and spinal fluid. There is a band of analgesia around his chest about 3 inches wide. There is no analgesia in the legs.

His *history* is as follows: He is thirty-seven years of age, single, a barber. He had a chancre three years ago. He had mercurial rubs on and off right along, but never for more than two weeks at a time, usually about one week, with intervals of a week or two or a month in between. Five or six months after the chancre appeared he suddenly had vomiting and cramps which were associated with a binding sensation around the abdomen. These pains would also come at intervals. Sexual and bladder functions were normal. He has been operated on, as you can see by these large abdominal scars. He has two scars, one large and the other smaller. He was operated, probably for root symptoms. He probably had one of these attacks of meningomyelitis which usually precede the other symptoms, and his abdomen was opened up because of the pain and vomiting.

Today I want to speak of the differential diagnosis.

Differential Diagnosis.—Tabes is not at all difficult to diagnose when it is well advanced. Probably the only condition which can simulate tabes at all successfully is combined degeneration. When combined degeneration is localized largely in the posterior columns, or before the degeneration becomes essentially combined, before you have both posterior column involvement and lateral column involvement, then the disease picture can very closely simulate tabes. In combined degeneration Weigert Pal stain may show a patch of degeneration occupying the posterior column. It may spare the gray matter, the fibers close to the posterior commissure, and the lateral fibers of the posterior column. Degeneration may appear in the posterior column and very little in the lateral column; then you have much the same pathologic picture as in tabes. The symptoms will be those of involvement of the posterior column, loss of joint and tendon sense, some anesthesia, some ataxia, and lost reflexes. As soon as the degeneration spreads to the posterolateral portion then you have a Babinski sign; that makes an entirely different picture. In the early stages you have to depend on two or three things for the differential diagnosis. First of all, in tabes it is very rare not to have a history of acute lightning pain. If you inquire carefully you will find that they are not cramps, whereas the pains which occur in combined degeneration are cramps and can be brought out in the history as cramps. A person goes to bed feeling perfectly well, and after he is in bed a while he has a cramp which pulls the leg up, and it will stay that way until he gets up and stands on it. This is essentially different from the sticking, jabbing pain in the calf of the leg which comes in tabes. So these pains versus the cramps help in the diagnosis. In the combined degeneration there is no Argyll Robertson pupil, but in early tabes there may be no Argyll Robertson pupil. The spinal fluid findings are essentially different in the two diseases. In combined degeneration there is rarely an increased cell count, a positive Nonne; the Wassermann is negative. In tabes the cell count usually runs from 10 to 150 per cubic millimeter. There is a positive Nonne and a positive Wassermann.

The blood-picture in combined degeneration is usually that of pernicious anemia, whereas any anemia in tabes is mild and of a secondary type. Then as the combined degeneration becomes established a Babinski sign appears, quite marked bilaterally. A Babinski sign is a manifestation of a disturbance of function of the pyramidal tract, and the pyramidal tract is not involved in tabes. Consequently, you do not get a Babinski sign in tabes, and there is no reason why you should get it. A Babinski sign always means upper motor neuron disease, and tabes is entirely a matter of posterior root and posterior column disease.

Multiple neuritis is another disease which is to be differentiated from tabes. In multiple neuritis the pains are quite comparable to the pains in tabes, for the reason that the tabetic pains are expressions of disease of the posterior root, namely, of the nerve. Consequently, in neuritis you would expect to get the same irritative phenomena that you do in the irritative stage of tabes. The pains are of a little different character and can be easily confused. There is loss of deep reflexes, especially in the ankle and the knee. In multiple neuritis, which is very frequently due to alcohol, there is a fixed pupil to both light and accommodation. It is not at all uncommon to see patients come into a hospital with a perfectly fixed pupil, but as they stay there long enough to get the alcohol boiled out of them the pupil begins to react to convergence, but stays fixed to light, becoming a syphilitic or Argyll Robertson pupil. But in multiple neuritis you get tenderness associated with the pain, whereas in tabes you get loss of sensation. If one squeezes the calf of the leg, it is very painful; if one tries for a Babinski sign and strokes the sole of the foot, it is very painful, whereas just the opposite is true in tabes.

So in the diagnosis between multiple neuritis or tabes that difference in sensation is the thing you want to look for.

Then in early tabes a primary optic atrophy has to be differentiated. That is the problem in the following case. There is primary optic atrophy here. Tabes or lues is the chief cause. In a boy of this age, nineteen years, with a primary optic atrophy, you have always to think of the sella turcica, so tabes always has to

be differentiated from hypophyseal disease. In this case the diminished pain sense, the diminished knee-jerk, the mental phenomena, and the laboratory tests are what we have to depend on in the diagnosis of tabes. The *x*-ray picture of the sella turcica will differentiate the hypophyseal disease. Primary optic atrophy can occur in hypophyseal disease, but it is the form which ordinarily occurs in lues. A secondary optic atrophy has large veins and small arteries. In this patient the vessels are very small. The arteries are very small and the veins are even smaller. In secondary optic atrophy we speak of the relationship of the size of the veins and arteries. The veins are larger than they should be in proportion to the size of the artery, whereas in this boy the veins are not larger than they should be for the size of arteries present.

Another point which comes up frequently is to differentiate the various symptoms of tabes from other diseases. More tabetics are being treated for rheumatism than anything else in the early stages, and because of the fact that aspirin given in large enough doses takes care of the pains in tabes as a usual thing, both the doctor and patient are satisfied that that is the correct diagnosis.

Many tabetics have been operated upon, laparotomies performed, more than anyone has any idea of. Gastric crises operated on as appendicitis or gall-bladder cases are very, very common.

Treatment.—Early tabes is unquestionably curable. It is just as curable as syphilis, because that is what it is, but it has to be kept after. If you are going to cure a tabetic you have to give him more treatment than it takes to cure a syphilitic in the early stages. You might say that early syphilis is syphilis of the blood-vessels and late syphilis of the parenchyma cells. Consequently, you have to saturate the individual more and more with medicine and keep him saturated over a longer period of time.

Every case should have inunctions of mercury, 1½ drams a day, for three or four days until they are saturated, and then keep them in that condition over a period of three to six months.

If you can get them to stick for six months, do it. As they rub with mercury they must take good care of their teeth. Dr. Patrick advises his patients to use powdered potassium chlorate 2 ounces, oil of cloves ½ dram, rubbed up together with care. If the trituration is not done carefully it is likely to explode. This should be used after each meal, the gums as well as the teeth being scrubbed with it. If this is used regularly all patients can stand 1½ drams a day, and the thing to do is to get as much of the drug into them as possible. If the teeth are not well cared for they will probably not stand ½ dram a day. After a good course of mercury, either by inunction or deep muscular injection, then you can consider giving them an arsenic preparation, either salvarsan or neosalvarsan, or arsenobenzol, or diarsenol, but it is best to give that in as large doses as they will stand, and give three to six intravenous injections at intervals of three to five days for the first two or three and after that at two-week intervals. All this time you must watch the kidneys, because the kidneys must excrete a tremendous amount of the drug. It is not at all uncommon after giving neosalvarsan to see albumin appear in the urine when you try to crowd potassium iodid on the patient. If you take away the potassium iodid the albumin will disappear, but if you try to crowd the kidneys after these large doses of salvarsan you will very often have trouble. After this treatment with arsenic you should go back to the mercury for two or three months, then some more arsenic, and then back to the mercury, keeping the treatment up for two or three years, and then a period of rest. When giving intravenous injections of salvarsan at the same sitting withdraw 15 to 20 c.c. of spinal fluid. There is an idea that because of that withdrawal of spinal fluid we get a little more effect of the drug.

This covers pretty well the essential treatment of syphilis. During the course of the mercurial treatment it is well to give potassium iodid, running it up to 30 or 40 grains a day, and then stopping it for a few days. If you have a small amount of albumin in the urine, and the iodid does not increase it too much, the drug will be taken care of, but if you see the albumin beginning to

increase and casts appear, and the patient begins to have headaches and his blood-pressure begins to go up, the dose must be diminished.

Tabetics are often disabled, and you have to take up some means of teaching them to handle themselves again, and for this Frankel's exercises are the best thing I know of. Frankel's exercises depend upon making the patient make every move in an exact manner and thereby teach his muscles to act in a systematic manner. Not much apparatus is required. One thing you need is a cloth, or else to have painted directly on the floor about twenty steps. The foot-prints are placed with the heel about 2 inches in advance of the opposite toe, and then the patient is taught to put his foot in the footstep just as outlined on the floor. This exercise should be done several times a day. Patients who cannot walk have to be started in even a more elementary way than that. Something can be hung over his bed, and he raises his legs so as to touch this object, and the legs should be raised in a perfectly symmetric way; he should not jerk the leg up, but move it as he would move it when he is walking. By carefully following Frankel's system of exercises a great deal can be done to re-establish the ability to control one's movements.

CLINIC OF DR. CHARLES LOUIS MIX

MERCY HOSPITAL

PRESENTATION OF A CASE OF CARCINOMA OF THE HEPATIC FLEXURE

Association of Edema, Ascites, and Abdominal Tumor. Absence of Pain Significant. Röntgenologic Evidence of Obstruction at Hepatic Flexure. Prognosis.

February 27, 1917.

THIS patient has the following history: She is fifty-one years of age, and entered the hospital complaining of a swollen abdomen and swollen ankles. In other words, the usual complaint of dropsy.

Previous Illnesses.—She had measles at three years of age and cannot recall that she was ever sick before or since. She was perfectly well until recently. This last statement is noteworthy. It excites one's surprise to have a patient enter with a swollen abdomen and feet enlarged from edema, saying that she was perfectly well until recently. Such a statement has actual diagnostic value.

Family History.—Her mother is living and well at ninety-five. Her father died at sixty-five, cause unknown. A sister and a brother died in infancy; she has four living sisters who are perfectly well.

Present Complaint.—About four months ago she noticed that her ankles were swollen at night, usually an indication of cardiac weakness of some sort. About February 15th her abdomen also began to swell. Subsequent to that date she began to have trouble with her stomach and vomited quite frequently, almost every other day. She also had some pain in the right lower quadrant of the abdomen which came on rather suddenly

one night about Christmas time; this lasted two days and subsided upon the application of a hot-water bottle. She has had no pain at all since that time.

The first point to be emphasized in this history is the edema of the legs and feet beginning four months ago, preceding the pain at Christmas. The second point to be emphasized is the pain at Christmas in the right lower quadrant. The third point is the swollen abdomen which appeared on February 15th, with a sudden onset. The pain at Christmas disappeared shortly and she has had none since; we may, therefore, conclude that whatever her trouble is, it is fairly painless. The fourth point is the vomiting, which began about Christmas and was worse during January. She has not vomited since she has been in the hospital except when she was given a bismuth and buttermilk meal.

She drinks tea and coffee in moderation; she never eats much meat; she has never had any urinary disturbances, does not have to get up at night, and the bowels are reasonably regular. There has been no trouble with the nose or throat, no trouble in her chest, and no shortness of breath.

The causes of edema are usually cardiac, renal, or hepatic. Cardiac edema is always associated with a good deal of dyspnea at one time or another. You may be very certain if you are dealing with cardiac edema there will inevitably have been a good deal of shortness of breath before the edema appeared, because the first sign of trouble on the part of the heart is shortness of breath. Why should a cardiac patient be short of breath?

STUDENT: Because of insufficient oxidation.

DR. MIX: That would not occasion a real dyspnea. The explanation is very simple. The lung is nothing but a multilocular elastic sac and is exactly comparable to a multilocular rubber balloon. When a balloon is distended it is not in elastic equilibrium, but when the balloon loses its air it comes back to elastic equilibrium. When we take a deep breath the lung is not in a stage of elastic equilibrium, but as we let it out it comes back to its elastic equilibrium. When a heart is diseased and not beating properly the elasticity of the lung is diminished by the resulting pulmonary congestion; therefore the individual with

cardiac disturbance will not get his air out of the lung because of the lost elasticity, and he will not get air in very well because insufficient air is passed out in expiration. Thus he will have hard work breathing, and the worse the heart trouble, the worse the breathing. The cyanosis does not come early; the cyanosis comes on late and increases the respiratory difficulty; but the thing that starts the whole process off is a loss of lung elasticity due to a moderate degree of pulmonary engorgement with blood. This patient has had no dyspnea, yet she has edema of the lower extremities and a fair amount of fluid in the abdominal cavity.

We next proceed on the idea that her edema is a renal affair; but this is unlikely because her edema first came on at night, and in the usual renal cases the edema first appears in the morning. The patients wake up all swollen up—feet, hands, and face; during the course of the day the swelling goes down and by night they look natural. In the nephritic cases the edema is generalized, although it is, of course, localized to some extent by the additional element of gravity in the legs when the patient is up and about, but the ordinary nephritic is swollen in his face and hands and in all of his subcutaneous tissues. In this woman's case there has been no generalized edema; it has all been in the lower extremities and the abdomen.

There is a third cause for edema in the lower extremities, and that is some obstruction to the passage of blood through the portal circulation. If there is something the matter with the ingress of blood into the portal vein, or if there is some obstruction in the branches of the portal vein after it enters the liver, there is apt to be an escape of fluid into the abdomen. It is a transudate and will ordinarily be straw colored, with not much albumin, a low specific gravity, and no cells suggestive of an inflammatory origin. It will contain no erythrocytes, no polymorphonuclear cells, a few lymphocytes (which are to be found in all fluids taken from the body), and that is all. Thus it is wholly unlike an exudate due to an inflammation of a serous membrane. An exudate contains much albumin, has a higher specific gravity, and contains numerous cells of distinctly an inflammatory origin. If an obstruction of the portal vein takes

place there always develops an ascites if the patient lives long enough. If the ascites is very marked it leads to a good deal of intra-abdominal pressure and encourages edema in the lower extremities, probably by preventing by pressure free return of venous blood through the vena cava inferior. You find that demonstrated beautifully in Pick's cirrhosis of the liver. In this you have an obstruction of the portal blood, the formation of ascites, and then later on edema of the lower extremities appears. So in her case we have to consider the proposition of obstruction to the flow of portal blood into and through the liver. Urinalysis shows a trace of albumin, a specific gravity of 1030, an acid reaction, no casts, rarely a red blood-cell, an occasional pus-cell, some epithelial cells present, a few crystals of oxalate of calcium—nothing which indicates any great involvement of the kidney. Another examination gave practically the same results—specific gravity of 1030, faintly acid reaction, no trace of albumin, no casts, an occasional red blood-cell, some epithelial cells and bacteria, and some mucus from the bladder. Nothing indicates nephritis sufficient to account for her symptoms. These are all the urinalyses which have been made between the 18th, when she entered the hospital, and the 27th. The blood-pressure also disclosed a state of affairs hardly compatible with a nephritis. The systolic pressure is 122, the diastolic 72. Such figures are not ordinarily obtained in cases of nephritis because that disease constantly raises the blood-pressure, while hers remains low. Even an acute nephritis lasting only a short time raises the blood-pressure moderately. Her low blood-pressure is good evidence that there is no nephritis in her case in any way capable of accounting for her condition.

Examination.—You will notice that she is a little thin and the fingers look a little drawn and rather pale. She has lost weight in the upper part of her body, but not in the lower part, so the sum total of her weight remains practically the same. Many people make a mistake in saying that they have not lost weight because in many instances with the destruction of tissue, water takes its place, and when the fluid is drawn off you find that they have lost 30 or 40 pounds. So it is with her.

In examining the patient we find that there is quite an accumulation of fluid in the abdomen. You notice the abdomen is protruding quite a good deal and that the navel is well flattened out. When we percuss the abdomen it shows a sort of resiliency and there is a wave spreading over it. I want to call your attention to the fact that there is no dilatation of the veins in the abdominal wall. You can tell from the tenseness, the feel of it, and the dulness on percussion that there is a great deal of fluid in the abdomen. We are going to take out some of this fluid and she will feel a good deal better. We took out some a few days ago and she felt very much better afterward.

You will also notice that the edema of the lower extremities is quite marked; the legs are somewhat cyanotic, they look as if the circulation in them was rather poor. The return of the venous blood is evidently delayed.

Right here let me say that there is absolutely no luetic element in her case. The Wassermann is negative and the clinical history is negative.

The outline of the abdomen is characteristic. Will one of you please come down and tell me what you notice on inspection? The thing to look for is symmetry—is this abdomen absolutely symmetric?

VISITOR: No, it is not; the left hypogastric region is dilated.

DR. MIX: That is, you mean it bulges more in that region so that the axis is diagonal from lower left to upper right. There is a lack of symmetry in the outline of the abdomen. An ascites pure and simple should not produce such an appearance. See if you can feel anything in the abdomen. We examined her when she came in and it was difficult to find anything because of the ascites. Yet we have determined that there was no enlargement of the liver from a hypertrophic cirrhosis, no real evidence of an atrophic cirrhosis, although it is true that the liver is pushed up by the fluid to a certain degree.

Now as to abdominal fluids. They are divided into exudates and transudates. The exudates are caused by an inflammation of some sort. The transudates are brought about by something which leads to a pouring into the abdominal

cavity of fluid from some surrounding part. The transudates usually show a low specific gravity; the exudates, a high one. The amount of albumin in the transudate is very slight, while in the exudate it is likely to be considerable. Exudates are usually straw-colored; some of them, however, are bloody. Bloody exudates are almost invariably due to one of two things, malignant disease or tuberculosis, though in the latter condition the amount of blood is often very slight.

(Intern makes abdominal puncture.) You will notice that as this fluid flows out instead of being straw-colored it is very dark red, and that it contains a good deal of blood. It is also very frothy because it contains a good deal of albumin. After we get most of this fluid out we shall be better able to palpate the abdomen. We have already done this, and we found that in the upper right quadrant there was a mass which was very vague in outline.

Abdominal masses are usually not sharply circumscribed, especially when they are on a malignant or infectious granuloma basis. You often can palpate a vague mass in the abdominal cavity, and all you can say is that you can feel something which has no well-defined edge. You can't tell where the mass begins and where it leaves off. These growths have a core, so to speak, and from that there radiates out a hardness which constantly diminishes in amount, until finally it shades off into tissue which feels perfectly soft. That is precisely the sort of mass we can feel in the right upper quadrant.

What can the mass be in this case? It must be something on a neoplastic basis, but where did it originate? Is it a renal neoplasm? No, because it has a vague outline, does not move sufficiently with respiration, and there is no blood in the urine except a few isolated corpuscles. Is this a retroperitoneal tumor which has invaded the peritoneal cavity? How can such a mass give rise to ascites? You will remember that the ascites came on rather abruptly on February 15th, so she has had it for only about twelve days, and yet this is the second time we have tapped it. It was also associated with other symptoms—pain at Christmas time and vomiting, indicating some disturbance

of the alimentary canal. You would not ordinarily expect in a retroperitoneal tumor such an occurrence as vomiting every other day, yet she had such vomiting. You would rather think it was something in connection with the alimentary canal. This mass seemed on palpation to be in the ascending colon at the hepatic flexure. It is entirely distinct from the edge of the liver.

The absence of pain in this patient at the present time is interesting. You will remember that her only pain came at Christmas when she overate, as I learned on cross examination. A carcinoma of the breast, for example, is usually absolutely free from pain at the onset. We have at present in the hospital a man with a large carcinoma of the stomach, and he assures us that he has had no pain at any time, and yet he has an enormous carcinoma which blocked the pylorus so that he had to have a gastrojejunostomy to keep from starving to death. Recall the painless carcinomata of the uterus in the early stages. In a woman like this, who has been perfectly well and who suddenly develops such an ascites and edema *without pain*, as this woman has done, we must always entertain the possibility of a malignant neoplasm of some sort.

We had a series of *x*-rays made and the plates are very instructive. (Exhibiting plates in shadow-box.) The first plate was taken about fifteen minutes after the bismuth was swallowed. The bismuth passed very rapidly through the wide open pylorus into the small intestine, and here you see a remarkable accumulation of bismuth in the small intestine within fifteen minutes of the swallowing of the meal (Fig. 202). The next picture shows the bismuth two hours later. There is still a great deal of it in the small intestine, but very little in the stomach, and here is a large dilated mass which contains air and no bismuth, which looks like a very much dilated cecum (Fig. 203). These two plates indicate that the bismuth does not easily and quickly flow into the large intestine, but stays in the small intestine. The third plate, taken five hours after, shows the bismuth in a mass in the small intestine, over to the right side. It looks like a bunch of California grapes (Fig. 204).

This last picture is illuminating. We gave an enema of

bismuth to find out how far it would go toward the small intestine, and here is this great mass of distended cecum into which the bismuth has not gone at all. In other words, right

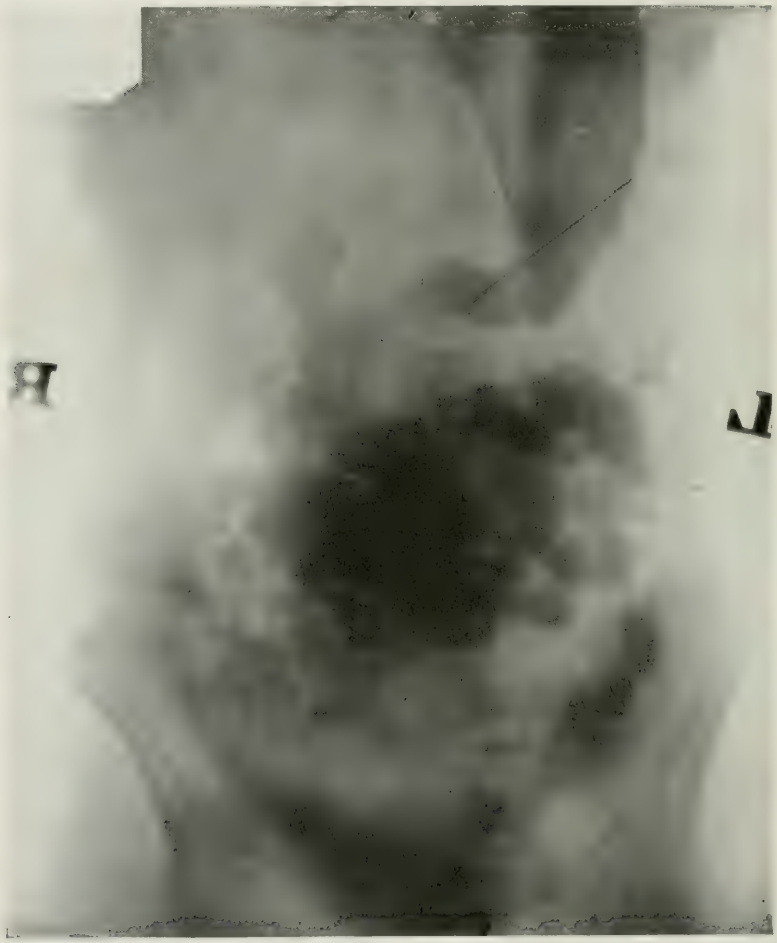


Fig. 202.—Fifteen minutes after ingestion of bismuth and buttermilk. Note the comparatively rapid emptying of the stomach and the retention of bismuth in the small intestine.

at that point is a blockade; the bismuth cannot get down from above and it cannot get up from below. *Note the diagnostic principle of dilatation behind the narrowing.* There is an accu-

mulation of gas behind the stenosis. If there is a blockade of the

Fig. 203.—Two hours after the ingestion of bismuth and buttermilk. Note the delay in the emptying of the stomach, the bismuth massed in the small intestine, and the gas in the cecum. Evidently a neoplasm has prevented the passage of the bismuth into the cecum. Since the neoplasm also prevents bismuth from passing into the cecum when given in an enema, an obstruction must be near the hepatic flexure and involve the terminal coils of the ileum.

pylorus the stomach will enlarge; if of the mitral valve the left auricle will enlarge, and if there is a stenosis of the hepatic

flexure of the colon there will be a distention of the cecum. Somewhere in the hepatic bend there is a blockade, and that is

Fig. 204.—Five hours after the ingestion of bismuth and buttermilk. Note the bismuth massed in the small intestine, giving an appearance like a bunch of grapes. The bismuth has been unable to make its way into the colon. Note the gas in the cecum.

where she had the pain when she overate on Christmas day and dammed up her food, and because of the blockade she vomited (Fig. 205).

The doctor has now removed at least 2 gallons of this fluid and she is much relieved in consequence. You will notice that

Fig. 205.—Colon after bismuth enema. It was impossible to get the bismuth into the cecum, which is much distended with gas. This is highly suggestive of a narrowing at the hepatic flexure, with a dilatation of the cecum behind the stenosed portion.

the abdomen is now flat, and you can now see a certain amount of *exaggerated peristalsis*, which is also an indication of obstruction in the alimentary canal. It is as if the coils of the small

intestine were pushing hard in an effort to push their contents past an obstruction.

(To the Student): Now see if you can feel anything in the abdomen. It is easy to feel a mass, which you could not do before, and, furthermore, you feel that it is *very hard*. Hardness, "woody" hardness, is one of the characteristics of a neoplasm of this type. The patient thinks that the color of the fluid is caused by the medicine she takes (Basham's mixture), and says that if it were due to blood she would be very much sicker than she is. She says that last summer she had a sensation "as if something did not go through," as if it got stuck and came back and she vomited it. She says that she feels very well now. (Patient removed.)

Now what is the diagnosis?

Visitor: A carcinoma of the large intestine located at the hepatic flexure.

Dr. Mix: Yes, and nothing can get down past that flexure and nothing can get up above it. This case illustrates diagnostic methods very well. At first sight this patient looks like a nephritic on account of the faint trace of albumin, edema of the legs, and ascites (such was the intern's diagnosis on his first visit), yet when you examine her carefully you find a bloody fluid, a mass in the abdomen, and a state of affairs which is very much worse than that associated with nephritis. What will happen if nothing is done? A complete obstruction will soon take place and then an artificial anus will have to be made. What is the use of waiting for that? Why not give her a certain amount of freedom by having something done operatively? I think a sensible thing would be to make an anastomosis of the ileum and the sigmoid, and if possible resect the carcinomatous portion of the colon. That, of course, will not relieve the carcinomatous peritonitis which is present, as shown by the bloody fluid, but its further development will be comparatively slow and she may live for several months.

This case has some interesting elements about it deserving comment. To me the matter of pain is very interesting, for she has had no pain except when she overate. Then the pressure

behind the blockade, with exaggerated peristalsis, led to pain. The *absence of pain* in a case of this sort always suggests the possibility of carcinoma. Many cases of carcinoma that I have seen have been without pain. We used to look upon pain in gastric carcinoma as a leading or cardinal symptom, but it is not. Pain is met with only in the stage of ulceration. About five years ago I had a patient with carcinoma of the ascending colon. His only symptom was obstinate constipation coming on in widely separated spells. His last serious blockade was relieved by croton oil. Strangely enough, in going through his early history there was complaint only of constipation. He had no pain until he got the pain of exaggerated peristalsis from obstruction, and when the blockade was raised the pain immediately stopped. There is no pain in carcinoma of the stomach or of the bladder in early stages. In papilloma of the bladder there is no pain until tufts of the tumor become lodged in the urethral opening, and then the patients have pain just as they would from a urethral stone, but they do not have pain because of the malignant papilloma. The absence of pain in cancers of the breast and uterus is noteworthy. When you see such a case as the one we have just shown, where grave pathologic processes are going on without pain, you should always wonder if it may not be malignant.

Another point of interest in this case is the accumulation of fluid. How did it happen? How did this woman happen to get edema of the legs and ascites? I think when her abdomen is opened, if it is done, it will be found that the vena cava is compressed directly or indirectly by the mass or by the associated carcinomatous peritonitis. In her case the first thing she noticed was swelling of the legs. I have seen patients with carcinoma of the breast stay away from physicians until the arms began to swell from lymphatic blockade. In this woman's case I think it is a venous blockade rather than a lymphatic blockade which causes the edema in the legs. You notice the peculiar mottling of the legs, showing interference with capillary circulation.

Why did the ascites develop? Is there any portal blockade

of the liver? Possibly the carcinoma lies close to the portal vein; it is either at or very close to the hepatic flexure of the colon, and, therefore, must lie close to the portal vein and to the transverse fissure of the liver. Is her portal vein blocked? I do not think so. Why? *In a portal blockade there is always evidence of a collateral circulation.* The venous blood must get up to the heart in some way, and one of these ways is by way of the hemorrhoidal veins. The superior and middle hemorrhoidal veins open into the inferior mesenteric vein and so send their blood to the portal vein. The inferior hemorrhoidal veins, on the other hand, drain into the internal iliac, and so send their blood to the vena cava inferior. Now when the portal vein is blockaded inferior mesenteric blood reverses its flow, passes down through the anastomosis between middle and inferior hemorrhoidal veins, and so to the internal iliac vein and vena cava inferior. Even blood from the splenic vein or the superior mesenteric may flow down through the inferior mesenteric from the portal vein, and so make its way to the inferior vena cava and into the heart. That is one of the ways that the blood gets around the blockade, and the result is a great enlargement of the veins around the rectum, with the production of hemorrhoids from this cause. Many of the cases of cirrhosis of the liver suffer from resultant hemorrhages from the rectum.

There is another possible anastomosis. If you will recall patients suffering from cirrhosis of the liver you will remember that many of them vomited blood. Why? The portal blood, brought by the splenic, inferior and superior mesenteric, and cystic veins, which ought to pass through the liver, flows backward through the gastric or coronary. An anastomosis is quickly developed between its venules and the esophageal veins, so that the portal blood makes its way to the azygos veins and so to the vena cava superior and to the heart. It is the rupture of the esophageal and coronary varices which leads to the gastric hemorrhage in cirrhosis of the liver, and the hemorrhage goes far to indicate the presence of such an anastomosis. We have no history of either hemorrhoids or the vomiting of blood in the case of our patient.

A third collateral anastomosis takes place between the deep and superficial abdominal veins. There is established a collateral anastomosis in the flanks shown by large blue veins streaming up toward the middle and anterior axillary region from the abdomen, permitting venous blood to find its way into the internal mammary, and so to the superior vena cava, and then into the heart. There are no such large and dilated veins in her flanks such as are present in almost all cases of cirrhosis of the liver. A fourth method of anastomosis is the rare one, where portal blood passes down the old embryonic vein from the liver to the navel, and then upward to the internal mammary. This demands a patency at least potential in this vein. Hence such an anastomosis (called the "caput medusæ") is very unusual, though it has a fatal facility for sticking in the minds of medical students. This patient has no *caput medusæ.*

In conclusion, then, in any case of portal blockade there is evidence of a collateral anastomosis, and such evidence clinches the diagnosis. This patient has no such evidence. There is no portal blockade. Evidently, then, the ascitic fluid comes from the carcinomatous peritonitis and she may be said to have a carcinomatous exudate. Many a malignant tumor pours out just this kind of a fluid into the abdominal cavity.

The *x*-ray plates illustrate the principle upon which you work. We first felt the mass and were convinced that it was in the ascending colon. The thing to do was to determine whether there was any blockade in the colon, and for this purpose there were two things to do. One was to send bismuth down from above, and the other to send it up from below to know whether there was any place where the bismuth was stopped. It stopped in both instances in approximately the same place. It would come down and fill the small intestine pretty well, but it could not get by the cecum and hepatic flexure of the colon. When it was put in from below it would pass upward freely into the sigmoid, the descending and transverse colon, but it could not get by the hepatic flexure of the colon into the cecum, thus proving that there is a blockade of some sort at that point.

CLINIC OF DR. FREDERICK TICE

Cook County Hospital

LUETIC INFECTION OF THE LUNGS

Pathology of Syphilis of the Respiratory Tract. Symptomatology and Physical Findings Not Characteristic. Points of Differentiation from Pulmonary Tuberculosis. Necessary Criteria for Establishing a Diagnosis.

Present History.—This patient is twenty-six years of age, and entered the hospital on January 21st with the following history: One week previously she noticed a feeling of numbness in the fingers of the right hand. After a couple of days the whole forearm and elbow became sore and swollen. She has occasionally had pains in the hips and in the chest, the pulmonary distress being aggravated by coughing. There is no history of a sore throat. She has been coughing considerably for several years and had pleurisy a few years ago. There has been no blood in the sputum at any time. She complains of weakness, loss of appetite, occasional sweats, and a slight loss of weight.

Past History.—She has had no previous similar attacks, was always delicate as a child, but had no distinct illness in childhood so far as she knows. One year ago she had pleurisy.

Family History.—Her parents are dead, cause unknown. She has one sister, living and well.

Marital History.—The patient is married; her husband is not well. She has been pregnant twice; miscarried at the seventh month with the first pregnancy and at the third month with the second. There is a history of a luetic infection for which she was treated for two weeks.

When she entered the hospital her noon temperature was

101.4° F., the pulse 120, respirations 32. In the afternoon of the same day it was 102.4° F., at midnight 102° F. The following day at 8 A. M. the temperature was 102.4° F., pulse 114, respirations 36. The noon temperature was 101.8° F.; 4 P. M. 101.6° F. On the 23d in the morning it was 100.6° F., in the afternoon 101.4° F. This record shows a morning temperature with an afternoon elevation. During the last day or two the temperature has not been much above normal. The pulse and respiration record has also gradually become less; pulse is recorded as 88 this morning, respirations 20.

Physical Examination.—From the general appearance of the patient we might conclude that she has lost some weight, but there is no evidence of a great loss; the skin and muscles retain a good tone.

The examination of the *chest* on inspection reveals little or nothing to attract our attention. The two sides are approximately equal; with ordinary breathing the expansion is equal. If there is any difference it is too slight to notice. With forced inspiration the expansion is about equal; possibly there is a little lagging on the left side in the infraclavicular region. The cardiac apex-beat is in the normal location.

On percussion there is impaired resonance above and below the clavicle on the left side. This extends well down to the interlobar fissure. On the right side above the clavicle there is impaired resonance, also present to a lesser degree immediately below the clavicle and extending downward to the second rib. Over the remaining anterior portion of the chest there is a good, normal resonance.

The right border of the heart is one fingerbreadth to the right of the right border of the sternum. The base is at the upper border of the third rib, but the cardiac dulness becomes continuous with the pulmonary dulness, so that it is not easy to determine the exact outline of the heart. The left border is in the midclavicular line.

On palpation, tactile fremitus is present over the entire right side, perhaps slightly increased above. On the left side over the upper lobe there is a very marked increase.

On auscultation, over the entire left upper lobe the breath sounds are roughened and accompanied by a great number of large, moist râles. Râles are also present over the lower portion, but they are not so numerous and there is no roughening of the breath sounds. In the midclavicular line, in the second intercostal space, the findings are suggestive of amphoric breathing. Over the right upper lobe the breath sounds are slightly bronchovesicular, accompanied by a few fine, crackling râles. Over the heart no murmurs can be heard. The second sound over the aortic area is accentuated.

In the upright position, on inspection posteriorly, there is little of note, but on percussion there is dulness over the left upper lobe, extending downward to approximately the interlobar fissure. There is an increased resistance over this area. Impaired resonance is present over the right upper lobe. Palpatory and auscultatory findings are the same posteriorly as indicated anteriorly.

Examination of the *abdomen* is negative. There is no enlargement of the spleen; the liver extends approximately a finger-breadth below the costal border. The kidneys are not palpable.

The *extremities* present a marked difference between the condition today and what it was a week ago. At that time the right elbow joint was perhaps three times as large as it is at present, with the maximum swelling at the joint, so that it presented a quite typical spindle-shaped joint. The joint involvement has rapidly improved during the past week, but there is still some swelling and tenderness, together with discomfort and pain on moving the joint. There is no crepitus, no roughening of the articular surfaces, and no indication of any fluctuation.

Inspection of the lower extremities reveals the presence of scars, more especially on the right leg, but also present on the left just below the knee, and on both of the thighs. These scars, the patient informs us, have been present since the occurrence of the ulcerations following her specific infection. The tissue-paper character of the scars with the pigmented borders is suggestive. The anterior border of the right tibia is rather irregu-

lar, perhaps somewhat thickened, as compared with the tibia of the left leg.

Fig. 206.—Skiagram of chest

The patellar reflex is somewhat exaggerated, as is the tendo achillis reflex. There is no actual involvement of the joints of the lower extremities, either the hips, knees, ankles, or smaller joints.

The glands of the neck are not enlarged; there is no demonstrable adenopathy in any portion of the body.

Examination of the eyes shows that the pupils are equal and respond promptly to light. There is nothing of importance in the mouth; the tonsils are enlarged.

Fig. 207.—Skiagram of right leg.

The Laboratory Findings.—The urine at the first examination showed a specific gravity of 1020, acid reaction, no albumin, no blood, and microscopically a few hyaline and granular casts.

The first white count made the day she entered showed 23,600, and a second count made yesterday showed 20,000. The differential count was normal. A Widal test was negative and the blood-serum Wassermann positive +++.

Repeated examinations of the sputum, particularly for

tubercle bacilli, have been negative. Pneumococci and Micrococcus catarrhalis were numerous.

Röntgen Findings.—To assist in the determination a Röntgen examination was made.

The report submitted by Dr. Blaine, the röntgenologist, is as follows: "The chest shows a high-grade fibrous condition of

Fig. 208.—Skiagram of right elbow

the lung areas; the left upper lobe appears to be consolidated. This dense shadow may represent luetic changes, while the balance of the shadows appear more like tuberculosis (Fig. 206). The shadows of the right leg (Fig. 207) appear not unlike those of lues; the left leg shows no changes. The right elbow (Fig. 208) is slightly less clear than usual, but no changes are noted."

DR. TICE: What is the diagnosis?

VISITOR: Probably a luetic involvement of the lungs or a tuberculosis with possibly a secondary pneumococcal infection.

DR. TICE: At the first examination the findings were suggestive of a pulmonary tuberculosis, and we expected to find tubercle bacilli in the sputum. But repeated examinations by the routine and the antiformin methods have revealed no positive findings. The resident physician has become somewhat discouraged from his repeated search with absolutely negative results.

The involvement of the elbow suggested the possibility of a tuberculosis, but on account of the acute onset, the inability to demonstrate tubercle bacilli in the sputum, and the absence of any definite change in the skiagram such a diagnosis was impossible. Due to the mono-articular involvement and because the patient has a slight vaginal discharge a gonorrheal arthritis was suspected, but the examination of the vaginal smears for gonococci was negative.

A third possibility was an acute arthritis due either to the streptococcus or the pneumococcus or some other type of organism. At no time were we able to demonstrate a sufficient amount of fluctuation or localized tenderness to justify a puncture of the joint with the object of obtaining fluid for diagnosis. Blood-cultures were made, but found negative.

The skiagraphic changes, the scars, the admitted luetic infection, and the positive Wassermann brought up for serious consideration the possibility of syphilis as the underlying cause not only of the osseous changes but also of the pulmonary condition. Is this a syphilis of the lungs, a pulmonary tuberculosis, or is there a combination of syphilis and tuberculosis?

Formerly, before the discovery of the tubercle bacillus, syphilis of the lung was not infrequently reported. Subsequently, when it was possible to demonstrate the existence of the tubercle bacilli in the sputum, many of the cases considered as luetic had to be pronounced tuberculous. Then for a period the diagnosis of syphilis of the lungs was rather infrequent, and has remained so until the organism of syphilis was

discovered and the Wassermann came into general use. Syphilis of some of the other internal organs, such as the liver, the gastro-intestinal tract, the cardiovascular system, especially the aorta and myocardium, is relatively frequent. At some time in the course of the luetic infection there is a diffuse systemic involvement and dissemination of the organism in various parts of the body which can be demonstrated at the time of the appearance of the various manifestations, or in the postmortem examination even years after the primary infection. If this is true, it is not unreasonable to suppose that in the secondary or the tertiary stage the lungs become infected and pathologic changes result from the syphilitic infection. An involvement of the respiratory tract is probably more frequent than is generally suspected. Luetic infection in the upper respiratory tract, such as mucous patches in the mouth, laryngitis, or bronchitis, is fairly common, and it is reasonable to infer that the lungs also become infected.

Pathology of Syphilis of the Respiratory Tract.—Let us first inquire what form syphilis may assume when the respiratory tract is involved. First of all, syphilis may occur in the respiratory tract as a congenital or acquired manifestation. In the congenital type of syphilis of the lungs the lesion may be circumscribed or diffuse. To the former belong the gumma and to the latter the pneumonias, either the white pneumonia of Virchow and Weber or the interstitial pneumonia. Congenital gumma of the lung is relatively rare, while a white pneumonia and interstitial pneumonia not infrequently occur in combination. The interstitial pneumonia is the most frequent congenital type, usually in combination with one of the other forms.

The acquired type is the one in which we are most interested, particularly in association with this case. In acquired syphilis the pathologic change may be limited entirely to the trachea, the bronchi, or the larynx, producing ulceration, cicatrization, and stenosis. A bronchiectasis or other serious conditions secondary to an obstruction may result.

Where the pulmonary tissue is involved the anatomic changes are variable and may present the following changes:

1. Gumma. A gumma in the lung tissue histologically is identical with the gumma in any other organ, and follows very much the same course as the hepatic gumma, that is, the tendency is not to go on to destruction, but to cicatrization. Consequently, this has some bearing upon the clinical findings. In the report of the röntgenologist there is no mention as to the appearance or existence on either side of a cavity. Gummata of the lungs have a tendency toward fibrous formation, just as occurs in the liver where the regular, stellate scars indicate the nature of the infection. In these organs it is the scar-formation that is indicative of the disease. If the process produces cavities they are usually small and limited as compared with the cavity formation in tuberculosis. Gummata of the lung, single or multiple, may occur in any portion, but more frequently involve the substance of the lung than the surface, particularly about the vessels and bronchi of the hilus of the lung, the lower lobe, and then the upper lobe, in order of frequency as given.

2. Luetic pneumonia of the lobular or bronchopneumonic type.

3. Fibroid induration—which may be most marked at the hilus, or occurs as separate, isolated areas or a diffuse involvement of an entire lobe or lung.

4. Syphilitic phthisis. In this type the lesion is progressive and destructive, presenting clinically many of the symptoms and signs of a pulmonary tuberculosis or phthisis. Perhaps a better and less confusing term would be "pulmonary lues" or "syphilis of the lung."

Symptomatology.—Clinically, the symptoms are variable, but a *cough* is usually present and is, perhaps, the first and most frequent manifestation. At first it may be due to the laryngitis or to a bronchitis, but later to the actual pulmonary involvement. *Dyspnea* is relatively frequent, variable in degree, depending on the severity of the infection and anatomic changes. Slight shortness of breath to paroxysmal attacks simulating bronchial asthma may occur. *Fever* is usually present, and in the more advanced cases is of the hectic type.

Hemorrhages are relatively infrequent, but may occur, usu-

ally as a late manifestation due to ulcerative changes. *Expectoration* at first is scanty, but later becomes profuse, fetid, and offensive.

Loss in body weight is absent or slight. In tuberculosis one of the clinical symptoms is loss in weight. In a luetic infection of the lungs this ordinarily does not occur, the weight is usually well maintained, and may be of some value in the differential diagnosis. *Pain* is usually not a marked feature, but *night-sweats* frequently occur in the advanced stages.

The **physical findings** are so closely identical and suggestive of pulmonary tuberculosis that a diagnosis is usually made of a pulmonary tuberculosis. Some writers have laid emphasis upon the location of the syphilitic involvement, maintaining that the lower lobe is more frequently involved than the upper, but this is hardly to be confirmed by the recent literature. It seems to be as frequent in the upper as in the lower, perhaps even more frequent at the hilus than in the apex or at the base.

In the definite determination of syphilis of the lung it is necessary, first of all, to exclude as far as possible a pulmonary tuberculosis. If the symptoms and signs are those suggestive of a pulmonary infiltration every effort must be made to exclude tuberculosis, and this means, in the majority of cases, repeated, thorough examination of the sputum. Although written some years ago, J. K. Fowler, in his discussion of the physical signs of pulmonary syphilis and tuberculosis, presents the subject with gratifying present-day accuracy. He directs attention to the following points:

"1. Tubercle usually affects the apex of the lung and subsequently the apex of the lower lobe, and tends to progress along a certain route. The primary lesion of syphilis is often about the root and central part of the lung; the disease follows no definite line of march, and gummata may be found in any position.

"2. Both tubercles and gumma may undergo either necrosis and caseation or fibrous transformation, but with caseous tubercle the tendency toward softening and cavity formation is the rule, whereas a caseous gumma very rarely breaks down.

"3. The progressive destruction of the lung by a process of disintegration leading to a gradual increase in the size of a cavity, a change so commonly observed in tubercular disease, is rarely if ever observed in syphilis except as a secondary result of stenosis of one of the main bronchi.

"4. In nearly all cases of advanced destruction of the lung occurring in the subjects of syphilis, stenosis either of the trachea or of one of the main bronchi is present, whereas this lesion is very rare indeed in tuberculosis.

"5. The cavities found in cases of pulmonary syphilis are usually bronchiectatic, but not invariably so; whereas in tuberculosis they are commonly due to progressive destruction of the lung, but may be bronchiectatic.

"6. The tendency to the formation of pulmonary aneurysms, which is so marked a feature in tuberculosis, is rarely observed in pulmonary syphilis.

"7. Pulmonary lesions in tuberculosis are very common, whereas in syphilis they are extremely rare."

Diagnosis.—In establishing the diagnosis of syphilis of the lungs it is necessary first of all to demonstrate absolutely the existence of the syphilitic infection either by the admitted history of the patient or by the presence of other syphilitic manifestations, either cerebrospinal, cardiovascular, osseous or hepatic, or by the demonstration of the specific organism or the presence of a positive Wassermann. To establish the diagnosis of pulmonary syphilis, referring again to J. K. Fowler's admirable article, the following conditions must be fulfilled:

"1. The cases must be complete, that is, the symptoms observed during life must be considered in connection with the lesions discovered on postmortem examination.

"2. The evidence of syphilitic infection must be undoubted.

"3. Repeated examinations of the sputum must have been made, and tubercle bacilli have been invariably absent, and the absence of tubercle from the lungs (as the cause of the lesions) must be proved by postmortem examination.

"4. Syphilitic lesions about the nature of which there can be no doubt must be found in other organs."

The Röntgen findings are of assistance in determining the presence or absence of tuberculosis or syphilis of the lungs. The röntgenologist reports that this is possibly a luetic involvement.

A still further proof, and one of considerable importance, is the influence of specific treatment. In the absence of a definite diagnosis active specific treatment should be instituted, and if improvement occurs it will establish confirmatory evidence of a luetic infection.

CLINIC OF DR. WALTER W. HAMBURGER

COOK COUNTY HOSPITAL

HEMATEMESIS

Presentation of Three Cases: Case I. Syphilitic Cirrhosis of Liver with Fatal Hemorrhage from Esophageal Varix. Autopsy Findings by Dr. H. G. Wells.

Case II. Hemoptysis Simulating Hematemesis.

Case III. Pernicious Anemia with Gastric Hemorrhages. Classification of the Causes of Hematemesis. Exulceratio Simplex of Dieulafoy. Frequency of Fatal Gastro-intestinal Hemorrhages Due to Cirrhosis of Liver.

Treatment: (1) Medical; (2) Surgical.

January 20, 1917, 11 A.M.

THIS patient, an ice-cutter, thirty years of age, while coming to Chicago from the country on a train, Monday, January 15th, complained of slight epigastric distress. On arriving in the city he ate a rather heavy dinner, consisting largely of beefsteak. One hour after his meal he became nauseated and vomited blood. Twenty-five to thirty minutes later he again vomited large amounts of bright red blood. He became extremely weak, but managed to leave the restaurant and return to his home. During the next twenty-four hours he complained of extreme weakness and nausea, but did not vomit. He was able to be up and about, but did not return to work. On Wednesday (January 17th) he noticed his bowel movements were dark red and bloody looking, and his weakness became so extreme that he fainted several times during the morning. In addition to the vomiting of blood he complained of a slight cough of about one week's duration, and upon admission to the hospital in an ambulance a diagnosis of pulmonary tuber-

culosis with hemoptysis was made, and he was admitted to the Tuberculosis Hospital.

Past Illnesses.—He had malaria while in Arkansas three years ago; had always been strong and vigorous; denies having had previous stomach symptoms of any description. No history of venereal disease.

Family History.—Mother and father dead of unknown cause. One sister and one brother living and well. One brother died of dropsy.

Habits.—He has been in the habit of drinking three or four whiskies and four or five glasses of beer daily. He does not use tobacco.

His weight has been constant, varying around 165 pounds.

Physical Examination.—Scalp, cranium, nose, ears, and eyes negative. Mouth: Three carious teeth. Pharynx: Negative. Neck: Negative. Chest: Large, well formed, and muscular.

Lungs.—Resonant throughout. No râles. No increased fremitus.

Abdomen.—Slightly distended. No signs of free fluid. Liver and spleen not palpable. No rigidity. In the upper right abdomen there is an increased resistance, suggestive of a mass which is very indefinite, lying under the right rectus muscle. It is slightly tender and suggests an enlarged liver, palpable colon, or a new growth. The remainder of the abdomen is negative.

On admission to the hospital his pulse was 108, respirations 20 to 22, and he had a slight temperature—99.4° F.

He is extremely pale. His skin, mucous membranes, fingertips, scleræ, etc., being almost colorless.

In view of the history and in the absence of positive lung findings he was transferred from the Tuberculosis Hospital to our Medical Service, with a diagnosis of acute gastro-intestinal hemorrhage. I first saw him at 5 o'clock Thursday afternoon. That night he had his third severe hemorrhage in the ward, vomiting about 1½ quarts of a bloody red fluid. His condition all day Friday (yesterday) was extremely critical. His hemo-

globin was 22, red cells 1,140,000, and leukocytes 62,000. The differential count showing 88 per cent. polymorphonuclears, 6 per cent. large mononuclears, 2 per cent. eosinophils, and 4 per cent. small mononuclears. The differential examination of the red cells showed a few small nucleated erythrocytes.

His urine showed nothing abnormal.

His blood-pressure was not taken.

His pulse yesterday was at times imperceptible, rapid, and thready, varying from 96 to 116; at one time 128.

In view of the continued bleeding, he was given two deep intramuscular injections of 40 c.c. each of whole blood taken from a healthy ward attendant.

When I first saw him yesterday afternoon he was rather somnolent and quiet. His pupils were small and he lay perfectly quiet and relaxed from the effects of the large amounts of morphin. His pulse at that time, although fairly rapid, was of a good quality and regular.

Examination, which was rather superficial because of the fact that we did not wish to disturb him to any extent, fearing a repetition of his hematemesis, confirmed the findings made in the Tuberculosis Hospital. There was a faint systolic hemic murmur at the base of the heart. Our diagnosis was an acute gastro-intestinal hemorrhage of unknown etiology, and he was placed upon the following orders: Morphin, $\frac{1}{4}$ grain, hypodermically every six hours; ice-coil to abdomen; nothing by mouth.

In spite of stimulation his pulse became more rapid and weak, and at 10.30 last night it was practically imperceptible. The nurse's record shows that at 1.40 early Saturday morning his condition was very poor and at 2.15 he died.

Permission for autopsy was obtained, and this afternoon at 2 o'clock Dr. Wells will hold a postmortem. As this case is the first fatal gastric hemorrhage that we have shown in the clinic I would urge all of you if possible to come to the autopsy to check up our clinical findings.

Differential Diagnosis.—Before going there it might be profitable to discuss briefly some of the possibilities in this case. Let us summarize our findings:

A young man in robust health is suddenly seized with a profuse hematemesis one hour after eating a rather heavy dinner. This hemorrhage is repeated within one-half hour, and within forty-eight hours from the onset he comes into the hospital exsanguinated, profoundly exhausted, with bloody stools, and a weak rapid pulse. In spite of complete physiologic rest he suffers a third profuse hemorrhage and dies within twenty-four hours.

(1) Of course, the first thought is that he has suffered with a latent *gastric* or *duodenal ulcer* which has thus announced itself with a sudden profuse hemorrhage. However, the complete absence of previous stomach complaints would speak somewhat against this, although ulcer of the stomach, as we have shown several times in this clinic, often remains latent for many years, so that the absence of stomach symptoms previous to this sudden hematemesis does not of itself entirely rule out chronic ulcer. He is in the ulcer age, but he has never had, so far as can be ascertained, periods of indigestion, nausea or vomiting, or anything suggestive of stomach disease.

(2) *Carcinoma of the Stomach.*—There is nothing in the history or examination suggestive of cancer. He is not in the cancer age; he has lost no weight; he is not cachectic; he shows no emaciation. There are no findings pointing to carcinoma of the stomach, and I think we can safely rule it out.

(3) *Trauma.*—He has suffered no injury, and there is no evidence of trauma, although, as you know, severe crushing, abdominal injury is sometimes responsible for gastro-intestinal hemorrhage.

(4) *Cirrhosis of the Liver.*—You will remember he is a rather heavy drinker, taking four or five glasses of beer and three to four whiskies daily for the past five or six years. Likewise, you will remember, that there was felt an indefinite resistance in the right upper quadrant of his abdomen corresponding to the liver or gall-bladder region. It is possible, therefore, that he has a cirrhosis or other disease of the liver with a resultant dilatation of blood-vessels (varices), either in the stomach or in the esophagus, as is so common in alcoholics, and that this fatal hemorrhage has been caused from a rupture of one of the dilated vessels.

(5) *Aneurysm of the Thoracic Aorta.*—It is not a very uncommon event to have a large thoracic aneurysm erode the esophagus and cause thereby a profuse hematemesis. However, we found no evidence of aneurysm, no substernal dulness, no tracheal tug, no difference in the radial pulses, no suggestion of syphilis. We therefore feel justified in considering this possibility very remote.

(6) *Banti's Disease.*—Primary splenomegaly or splenic anemia with secondary cirrhosis is not frequently associated with gastro-intestinal hemorrhage. While it is possible that he has a Banti's disease, his spleen is not palpable or apparently enlarged. There is no ascites. He has no symptoms of a long-continued splenic anemia.

(7) *Syphilis of the Stomach.*—Syphilitic ulcer or gumma is not infrequently a cause of gastric hemorrhage, particularly where the syphilitic process, as is so frequent, has invaded the blood-vessels, producing endarteritis and arteriosclerosis. If one can rely upon his history the probability of syphilitic disease of the stomach is ruled out, with the possible exception of a congenital syphilis.

(8) *Hemorrhagic Erosions or Acute Hemorrhagic Ulcers.*—Dieulafoy first described this rather obscure condition under the name *exulceratio simplex* as follows:

"A man twenty-seven years of age was admitted under my care; his pallor at once pointed to severe hemorrhage; he had had several attacks of hematemesis and passed much blood per rectum; he put the quantity of blood vomited at 3 pints. Although the classical signs of gastric ulcer were present, I thought of a latent ulcer which had perhaps opened a large vessel. In spite of treatment the bleeding recurred during the night. The next morning two basins were nearly filled with blackish blood and clots. At one o'clock in the afternoon fresh hematemesis and death. He had lost about 7 pints of blood in less than thirty hours.

"Here are the results of the autopsy: The exterior aspect of the stomach was normal. To our great surprise, we did not find a simple ulcer. The mucous membrane was carefully

washed, and we then discovered ½ inch from the cardia a superficial circular *exulceratio*. It was so superficial as to affect the tunica mucosa with its muscularis mucosa. At the bottom of the ulcer we saw the gaping arteriole which caused the fatal hemorrhage."

He quotes several other similar cases, and concludes that the clinical history of *exulceratio* commences with hemorrhage; that hematemesis is generally the first symptom; and that it is usually massive or fulminant in type.

While it is possible that our patient suffered with a hemorrhagic erosion, the presence of the mass in the right side and the history of alcoholism suggests a more likely cause.

(9) *Disease of the Nasopharynx, Mouth, Gums, Etc.*—Dilated vessels, quinsy sore throat, abscesses, small aneurysmal dilatations in this area may account for sudden hematemesis or epistaxis, and must be thought of in every case from the standpoint of differential diagnosis. Routine inspection, as well as could be made in the ward in this patient, showed no evidence of disease of this type.

(10) *Hemoptysis, or the Bringing Up of Blood by Act of Coughing from the Lungs, Bronchi, or Trachea.*—It is often rather difficult to distinguish between hemorrhage of the respiratory tract and the gastro-intestinal tract, as this case well emphasizes. In spite of bearing this possibility in mind, and of extreme care in the obtaining of a history and the examination of bloody fluids, mistakes are often made. Within the past eighteen months I have had two experiences of this kind. One where a hematemesis was mistaken for a hemoptysis and the other just the reverse. The complete absence of chest findings and the definite statement of the vomiting up of blood in our patient makes it fairly certain that the source of the blood should be sought for in the gastro-intestinal rather than in the respiratory tract.

(11) *Pernicious Anemia.*—Gastric hemorrhage is not entirely unknown in this disease. This is also true of the leukemias and some of the purpuras, but in our patient there has been nothing

to suggest the likelihood of blood disease, with one possible exception of the rather high-grade leukocytosis (62,000).

In a general way this covers the differential diagnosis of gastro-intestinal hemorrhage. As far as the direct diagnosis in this particular case goes, while an absolute diagnosis probably should not be attempted because of the short period of observation of the patient and the necessarily superficial examination, it would seem fair to conclude that in all probability he suffered from one of two conditions—either a latent ulcer with a sudden acute fatal hemorrhage or the hemorrhagic erosion of Dieulafoy, or from dilated varices in the esophagus secondary to cirrhosis of the liver. If one were forced to choose between these two merely on the findings one might be swayed somewhat to the latter, in view of the history of alcoholism, the resistant-like mass in the right upper abdomen, and the absence of gastric symptoms pointing to chronic ulcer.

January 20, 1917, 2 P.M.

Postmortem by Dr. H. G. Wells, of Cook County Hospital

External Appearance.—The body is that of a powerfully built white man, well nourished, but extremely pale. Rigor mortis is well developed. There are numerous brown pigmented moles scattered over the body. The sclera is white. The mucous membrane is extremely pallid. The superficial lymph-glands are not enlarged. There is no edema. The nail on the right great toe is fractured longitudinally and greatly thickened. There is blood on the adjacent toe, but on examination does not appear to have come from the great toe. The buttocks have a yellowish-green discoloration. (This is the site of an injection of blood.)

Abdominal Cavity.—There is about 5 mm. of subcutaneous fat. The inguinal and femoral rings are closed. The peritoneal cavity contains about 100 c.c. of a clear watery fluid. The vermiform appendix is embedded in adhesions posterior to the colon. Some loops of bowel are dark colored due to blood in the lumen. The peritoneal coats are smooth and shining.

The mesenteric lymph-glands are not enlarged. There are a few fibrous adhesions about the spleen. The margin of the liver is about two fingerbreadths above the costal margin.

Pleural Cavity.—In the left pleural cavity there are dense fibrous adhesions over the upper lobe. The lung collapses well and there is no fluid. The right pleural cavity is free from adhesions and fluid.

Pericardial Cavity.—The pericardial cavity contains about 50 c.c. of clear fluid.

Mouth and Pharynx.—Not examined.

Larynx and Trachea.—There is a little bloody mucus on the mucous surface of the trachea.

The *esophagus* is very much enlarged and dilated. On opening the esophagus the mucous membrane near the cardiac orifice is seen to be blood-stained and the stomach contains a large quantity of blood. There is a raised area about 1 cm. in width beneath the lower posterior part of the mucous membrane. This extends longitudinally, and on further examination is found to be a tremendously enlarged varicose vein, which has opened about 5 cm. above the gastric junction through a round perforation between 0.5 and 1 mm. in diameter, at which point there is no thrombosis (Fig. 209)

Thyroid and Thymus.—The thymus is completely involuted.

Heart, Aorta, and Vessels.—The heart is of about normal size, weighing 300 grams. The mitral and tricuspid orifices are normal. The valves are all normal. The myocardium is normal. The entire aorta is free from sclerosis.

Lungs.—There is a marked difference between the two lungs. The right lung does not collapse so well as the left. It weighs 800 grams. The lower lobe and the posterior part of the upper lobes are much darker than the remainder of the lung. These parts have a boggy feeling. The pleura is normal over them. The lower lobe is full of fluid that pours out on being squeezed. The main bronchi contain a frothy watery fluid. The left lung weighs 480 grams, has no areas of consolidation, and appears quite normal.

Peribronchial Glands.—The peribronchial lymph-glands are

not enlarged, contain only a small amount of coal pigment. There is caseation of a few small glands at the root of the left lung.

Fig. 209.—Esophagus showing dilated varix and point of rupture in syphilitic cirrhosis of the liver.

The *liver* weighs 1500 grams. It is remarkably deformed. There are dense adhesions between it and the diaphragm over the dome. There are firm fibrous adhesions between the gall-bladder and the mesocolon. The surface of the liver is very nodular and irregular. The right lobe is not a great deal larger

than the left. There is a tremendous enlargement of the Spigelian lobe, it being as large as the left. The Spigelian lobe is less

Fig. 210.—Syphilitic cirrhosis of the liver.

irregular and less nodular than the remainder of the liver. The quadrate lobe is also very much enlarged. The gall-bladder is of

normal size. The liver is hard and lumpy and is divided up into areas of 4 to 15 mm. in diameter by bands of connective tissue. The cut surface is also divided up into coarse lobules about 1 cm. in diameter. There are many brownish in color, although some

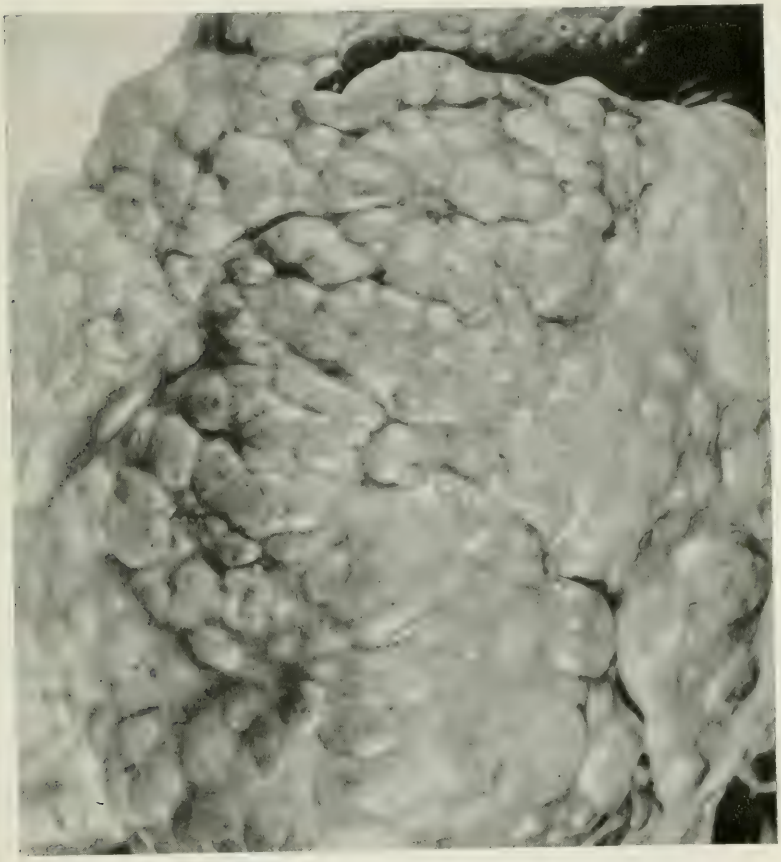


Fig. 211.—Syphilitic cirrhosis of the liver ("hepar lobatum").

are bile stained. In one section there is a well-defined white nodule 7 mm. in diameter (Figs. 210, 211).

The *spleen* is greatly enlarged, weighing 620 grams. It is symmetric and has a somewhat lobulated external surface. It is a little harder than normal and feels slightly nodular. The cut surface, however, shows no nodules, but there is an in-

crease in fibrous tissue. The lymph-glands about the hilum are very hyperemic.

The *pancreas* seems normal. The lymph-glands are hyperemic.

Gastro-intestinal Tract.—The stomach is almost entirely full of blood, but otherwise is normal. The intestines contain bloody material in some loops and the rectum is full of black, bloody material. The appendix is bound down by fibrous adhesions.

The *adrenals* are both small. They have a very thin cortex and are poor in lipoid content.

The *kidneys* are somewhat enlarged and weigh 400 grams. The cortex is thickened, but the cortical markings are well preserved. The capsule tears readily and the underlying surface is apparently normal.

The *urinary bladder* appears normal.

Generative Organs.—The testicles, prostate, and seminal vesicles all appear normal.

Lymph-glands in General.—The lymph-glands generally appear normal, except that they are somewhat hyperemic.

Brain, Meninges, and Spinal Cord.—Not examined.

Histologic Report

Liver.—There is a great increase in connective tissue, which divides the liver into lobules of varying sizes. It is not evenly distributed—some lobules are not affected. There is a round-cell infiltration, but not of the inflammatory type. The capillaries are much distended and there is apparent proliferation of bile-ducts. At one edge of the liver there is a horseshoe-shaped area of necrosis surrounded by a layer of polymorphonuclear leukocytes. This area resembles an infarct and shows no evidence of syphilis (Figs. 212–214).

Adrenal.—Normal, although rather poor in lipoid.

Peripancreatic Lymph-glands.—The sinuses are greatly distended with erythrocytes, but they are not hemolyzed.

Kidney.—The glomeruli are rather cellular and there are a few fibroid glomeruli. There is a slight precipitate in the tubules.

Testicle.—Normal and spermatogenesis is present.

Lung.—Normal except for a slight edema.

Pancreas.—Normal.

Spleen.—There is a marked fibrosis throughout, but especially in the subscapular region. The trabeculæ are especially marked.

Fig. 212.—Syphilitic cirrhosis of the liver (60 diameters). Great increase in connective tissue dividing liver into various sized lobules.

Anatomic Diagnosis.—Syphilitic cirrhosis of the liver. Hepar lobatum (healed congenital ?). Focus of anemic necrosis in liver. Compensatory dilatation of the esophageal veins with recent perforation into the esophagus. Blood in the stomach and intestines. Severe anemia. Chronic passive congestion and fibrosis of the spleen. Hypostatic congestion and edema of the right lung. Adhesive left fibrous pleuritis. Slight hydroperitoneum. Caseous tuberculosis of left peribronchial glands.

January 24, 1917.

I wish to discuss with you for a few minutes this morning the case we saw last week and which you followed at autopsy. I think you will agree that this was a most unusual and interesting case. It brings up so many points for discussion that it is difficult to decide where to begin and what to say. I shall therefore speak very briefly of just a few points.

Fig. 213.—Syphilitic cirrhosis of the liver (21 diameters). Area of anemic necrosis (infarct?).

While syphilis of the liver is not at all a rare condition, a congenital hepatic syphilis occurring in an adult, as Dr. Wells takes this to be, and resulting fatally from an acute esophageal hemorrhage, is a most unusual clinical and pathologic picture.

Pathology.—Hoppe-Seyler, in "Die Krankheiten der Leber," speaks of the great variability in the location, form, and dis-

tribution of the interstitial hepatic changes due to syphilis. Common to all syphilitic processes in the liver is the formation of large quantities of cellular connective tissue, with frequent transformation to firm scar-tissue. The interstitial growth is primarily interacinous, is in general highly cellular, and, similar to the alcoholic cirrhosis, may lead finally to atrophy. It may

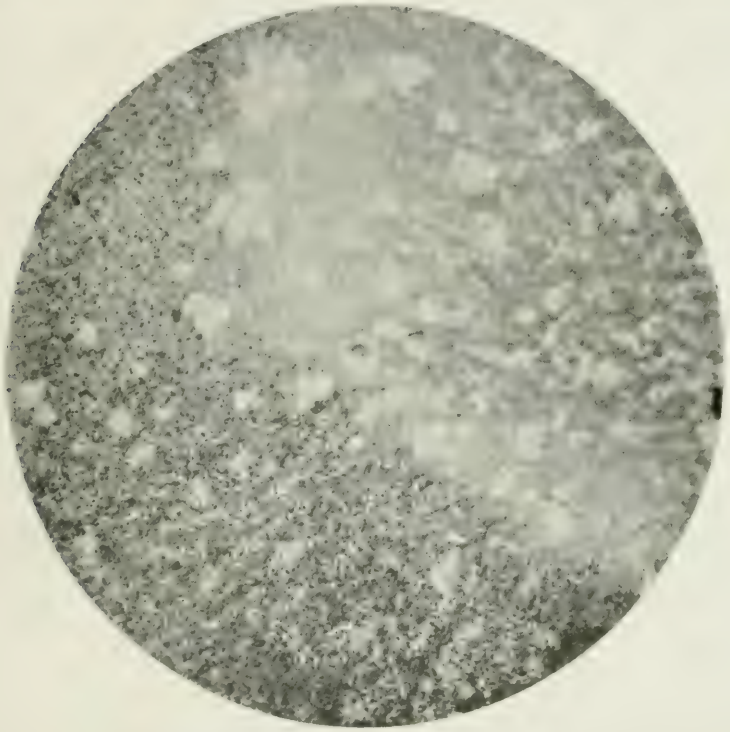


Fig. 214.—Syphilitic cirrhosis of the liver (110 diameters). Area of necrosis with surrounding layer of polymorphonuclear leukocytes.

result in a diffuse interstitial hepatitis, in the formation of single or multiple, large or small gummata, or in the production of an irregular, nodular liver, with particularly marked irregularities along the free liver edge, the so-called *hepar lobatum.*

The **clinical picture** of syphilis is likewise a bizarre one. The liver usually may be made out to be enlarged, it may be smooth, but more often irregular; it is often tender to pressure,

sometimes spontaneously painful. Icterus is usually absent or very slightly present. The spleen is usually enlarged and firm. Remittent or intermittent fever is not an infrequent accompaniment. From the atrophy there may result an ascites and other evidences of stasis, particularly when the syphilitic scar-tissue compresses the branches of the portal system. Often the splenic tumor assumes tremendous proportions, so that the picture closely resembles that of splenic anemia, pseudoleukemia, or Banti's disease.

Hemorrhages from the compressed veins are not infrequent, but, on the whole, less common than in the non-specific cirrhosis.

The general health of the patient suffers, resulting in weakness, pallor, cachexia, while recurrences of fever and chills may remind one of a sepsis.

The question of hematemesis in liver cirrhosis is a particularly interesting one. Here at the County Hospital probably a large number of the severe gastro-intestinal hemorrhages, because of the frequency of alcoholic cirrhosis, take their origin from dilated varices either in the esophagus or stomach. Dr. Preble[1] has collected statistics on 60 cases of fatal gastro-intestinal hemorrhage due to cirrhosis of the liver. His conclusions are as follows:

(1) Fatal gastro-intestinal hemorrhage is an infrequent and rare complication of cirrhosis of the liver.

(2) In the great majority of cases the cirrhosis is atrophic, but it may be hypertrophic.

(3) In one-third of the cases the first hemorrhage is fatal; in the other two-thirds the hemorrhages continue at intervals over a period varying from a few months to several years, the maximum given being eleven years.

(4) In one-third of the cases the diagnosis can be made at or before the time of the first hemorrhage. In the other cases the diagnosis cannot be made at all or only after months or years, during which time other symptoms of cirrhosis have developed.

[1] "Conclusions Based on 60 Cases of Fatal Gastro-intestinal Hemorrhage Due to Cirrhosis," by Dr. R. B. Preble, Amer. Jour. Med. Sci., 1900, vol. 119, p. 263.

(5) Esophageal varices are present in 80 per cent. of the cases, and in more than one-half of this 80 per cent. the varices show macroscopic ruptures, and it is probable that many other ruptures would be found if the varices were tested by injection of air or fluid.

(6) Fatal hemorrhages occur in cases which show no esophageal varices, and they are probably due to the simultaneous rupture of many capillaries of the gastro-intestinal mucous membrane.

(7) The hemorrhages in this class of cases are usually preceded by other symptoms of cirrhosis, but the first symptom may be a fatal hemorrhage.

(8) In 6 per cent. only of the cases which show esophageal varices was the cirrhosis typical; *i. e.*, showed ascites, enlarged spleen, subcutaneous abdominal varices.

Several of Preble's collected cases gave a history of syphilis, but none of them showed positive evidence of a syphilitic cirrhosis, so that from this standpoint our case is unique. In 42 cases in which the esophagus was examined varices were present in 35; that is, in 85 per cent. Preble makes several interesting observations as to the immediate cause of the hemorrhage, showing that "any sudden increase in the obstruction to the return flow of the blood may cause rupture. In several cases the hemorrhage was preceded by sudden and extreme muscular exertion, and in others by thrombosis of one or more of the main portal veins. In others the hemorrhage followed shortly after paracentesis. Possibly some of the cases were due to mechanical injury to the varix by some rough bit of food." The history of our patient suggests the possibility of such a cause.

Treatment.—Before going into the details of treatment, we might say that one should attempt to produce a thrombosis in the bleeding vessel. However, there are certain factors which may tend to prevent clotting. First, gastric peristalsis; second, the corrosive action of the gastric juice; third, possibly also increased blood-pressure.

(1) First, the patient is put to bed, absolutely quiet, in a darkened room with good ventilation, free from noise.

(2) Then morphin in adequate doses, allowing sufficient to give him absolutely mental and physical quiet. Morphin does several things: First, it controls him physically; second, it cuts out sensory stimuli; third, it probably has some beneficial effect on the stomach itself, causing increased tone.

(3) Cold is applied to the stomach and the abdomen, either in the form of an ice-bag or cold-water coil. It probably does no harm unless the cold is disagreeable to the patient. If the patient becomes restless, it should be discontinued. Recently some work has been done on the effect of external heat and cold upon the temperature within the stomach, and most of the observers think that cold does lower the temperature of the stomach slightly. From this standpoint it probably is worth while.

That is all; probably all that should be done for the first twenty-four hours. If he is doing well at the end of twenty-four hours he can have some nutrient enemata.

Now what are the dangers of this method of treatment? One is the production of acidosis due to the absence of food. Another is the development of parotitis, which occurs in individuals from an ascending mouth infection, from lack of chewing, and the accumulation of saliva. So acidosis and parotitis are the only dangers outside of increasing weakness and death.

Nutrient Enemata.—Of the various methods of rectal feeding, tap-water is to be preferred. The tap-water is rapidly absorbed. In my own practice I give 8 ounces of tap-water, to be retained per rectum, every four hours or four times a day. Or if you choose you can give it by the drip method if the patient will tolerate it. The thing to do is to combat the acidosis. On several occasions I have used 5 to 10 per cent. of pure alcohol in water. Alcohol is absorbed and has a high caloric value, and is of use particularly in acidosis.

Alcohol may set up some peristalsis and, therefore, may act in an undesirable way. The same applies to giving anything by mouth. Many men allow ice pellets, but my own feeling in the matter is that the ice simply dissolves in the mouth, water passing into the stomach and setting up peristalsis.

Now what else can be done in a medical way? If for any reason you believe that the patient is a hemophiliac you may want to give something to increase the blood coagulation time. The method we used yesterday was to give whole blood. Where you have a sclerosed vessel, nothing will do but either excising the bleeding point or tying off the blood-vessel itself, the same as controlling hemorrhage in any other part of the body.

That brings up the question of the relative value of medical and surgical treatment. Everyone agrees that most cases will stop bleeding if you leave them alone. That is, most gastro-intestinal hemorrhages stop and the patients survive if they are treated medically in this way without surgical interference.

There are two difficulties in the way of surgery. You often cannot find the bleeding point, or the patient is so anemic and so weak that he won't stand the slight additional shock necessary to the operation, even if the operation is done with local anesthesia. I have seen several patients die on the operating-table unable to stand the rise in blood-pressure and the operation itself. So, I say, there is an agreement that 80 or 90 per cent. of all these cases if left alone, under medical treatment, will get well, and the other 10 or 20 per cent. which die would die anyway with surgery. So the outlook in these cases is, of course, extremely bad.

There is some difference in the mortality statistics as relates to the age; that is, severe hemorrhage occurring in young people up to thirty and forty gives a better prognosis than in older individuals.

This concludes what I have to say on gastro-intestinal hemorrhage. I hope I may have aroused your interest in the subject to the extent that when face to face with the clinical problem itself some of the things we have spoken of here may recur to you. While possibly the most frequent cause of sudden hematemesis is chronic ulcer, as you have seen in this case, the hemorrhage was caused by a ruptured varix in the esophagus secondary to cirrhosis of the liver. However, if in the next case of hematemesis you allow yourself to be swayed by the experience of

this case and diagnose esophageal varix you would probably overlook an ulcer, and vice versa. So that it is only with a large experience and a careful examination and interpretation of the individual case that one can hope to arrive at the correct conclusion.

There are some further things of interest in connection with gastro-intestinal hemorrhage. For instance, Capps, at the Eighth Annual Meeting of the American Society for Clinical Investigation last year, reported 5 cases of acute hemorrhagic erosions of the stomach, studied clinically and bacteriologically. "In all 5 cases profuse hematemesis was the first symptom; in 4 cases there was some fever; in all cases there was evidence of associated infection during the period of treatment, namely, acute arthritis, lumbago, pneumonia, and herpes zoster; in all cases a primary focus was located, twice an acute tonsillitis, once a purulent rhinitis, and twice an alveolar abscess. In 1 case death occurred and multiple hemorrhages and ulcerations were found postmortem. Streptococcus was obtained from all the primary foci of infection. The conclusion is drawn that acute hemorrhagic erosions and ulcerations may often be recognized clinically, that they are of infectious origin, and frequently at least streptococcus is the causal organism."

January 27, 1917.

I want to show you these 2 cases in connection with the case that we discussed last Saturday because they bring up several other points in the question of hematemesis.

CASE I.—This is a dispensary case that I first saw November 3, 1915—that is, a little over a year ago. He is forty-six years of age. He came to the dispensary on account of vomiting, saying that he had stomach symptoms for three weeks at that time. In addition, he complained for three weeks of abdominal pain occurring from one-half to one and a half hours after meals, which was a rather severe sticking pain somewhat localized to the left side of his upper abdomen. The vomitus consisted of food eaten at that particular meal and came on one-half to one and a half hours after meals, the same as the pain, and *frequently*

contained dark red blood. He said he thought he vomited blood at least once a day.

In addition to the vomiting and pain he complains of nausea, anorexia, loss of appetite, and loss in weight (15 pounds in the last two months).

Examination at that time, over a year ago, showed nothing strikingly wrong with his abdomen, but quite a bit of tenderness in the epigastrium.

A single Ewald test-meal showed 100 c.c. of contents, free acidity of 33, and total acidity of 47, and no blood at the time of the examination.

The only suggestive thing in his past history is that at the age of eight years he was said to have pulmonary tuberculosis and coughed up blood; that is, he had a hemoptysis. On close questioning it was found that he vomited blood and did not cough blood. He was sent to the hospital last January and put through the usual examination, but nothing more could be determined unless he submitted to an exploratory laparotomy for further examination of the stomach and duodenum. He refused operation, and left the hospital in February and has been well up to the last two or three weeks of this year, when he again began vomiting and coughing up blood.

Now the question is, Has he had a real hemoptysis; has he a hematemesis, and, if so, what is the source? Is the blood from the duodenum or stomach? Is it from a duodenal or gastric ulcer or some other source of bleeding in the stomach , duodenum, or esophagus, or is it swallowed blood from his hemoptysis?

He has lost altogether about 20 pounds in the last four or five years. He has a cough at the present time. He is running no temperature. If we can get him into the hospital we will study him together and come to some definite conclusion.

Now on examining his chest he has in the right apex fine crepitant râles on deep inspiration; a little more dulness in the right scapular region and more dulness over the entire right chest than on the other side.

If you listen here, particularly in the right apex, you will hear these crepitant râles, and there is no doubt that he has a

pulmonary lesion. The question is whether the blood is solely from the lung or whether he has, in addition, a gastro-intestinal lesion. He has more findings in the right apex than the left.

His sputum has been examined several times without a positive finding.

Wassermann test is negative.

His abdominal examination is practically negative. He holds his abdominal muscles quite rigid, and it is impossible to examine him with any degree of accuracy. The spleen is not palpable, but there is a little increased dulness in the upper right quadrant, although no definite palpable mass.

Now, as I say, our interest in this case is purely the question of the *source of bleeding*. I wish to emphasize the point we made last time, the frequent difficulty of determining the source of bleeding, whether from the gastro-intestinal or respiratory tract.

CASE II.—This patient brings up some other points in regard to hemorrhage:

He is forty-eight years of age, Polish, and came into the hospital on January 11th with a diagnosis of "pernicious anemia." The history reads as follows:

Admitted for the second time January 11, 1917, he had originally come to the hospital August 28, 1916, with a history of illness of some eight months' duration. He then complained of anorexia and general malaise, loss of weight, noise in the ears, vertigo, dyspnea on moderate exertion, and a history of *two hemorrhages* from the mouth. There was no history of cough, but a history of night-sweats.

On *examination* he presented a marked jaundice, paresthesia of hands, feet, and toes to a marked degree, and also paleness of all mucous membranes.

Lung findings were negative. Sputum was negative.

Spleen was enlarged.

The *blood* presented a typical picture of pernicious anemia: Hemoglobin, 20 per cent.; erythrocytes, 1,276,000; white cells, 3000, besides many microcytes, macrocytes, megaloblasts, and poikilocytosis.

Under ascending doses of Fowler's solution and iron the hemoglobin went up to 80 per cent., erythrocytes went up to 3,392,000, and the general condition was improved.

He was then discharged on October 15, 1916, in fairly good condition. But now he returns again complaining of some abdominal distress, weakness, jaundice, and loss of weight. The blood-picture is that of pernicious anemia. The physical findings are the same as on the previous examination.

On January 22, 1917, the patient's general condition is about the same. A single Ewald meal showed 3 ounces of well-digested material, no free acid, a total acidity of 5. Examination of his stool shows a somewhat constipated one, of a light yellowish-brown color.

Now his examination is very interesting. We have not the time to go into it very completely, but we will speak of the things of greatest importance.

The first striking thing is, of course, the extreme pallor. The *skin* contrasted with normal skin is pale, and, in addition, is somewhat grayish yellow or light yellowish brown. It is not only pallor, but pallor associated with a yellowish tint. Notice the pallor of the finger-nails. His eyes and the gums are extremely pallid, as well as the lips and ears.

His pulse is somewhat rapid, but regular, of pretty good quality.

The *chest* has very good resonance, almost hyperresonance. He has no substernal dulness. I should have said that in addition to his pallor and the yellowish-brown discoloration he shows evidence of loss in weight.

Heart.—His cardiac borders are not enlarged. There is a very soft systolic murmur at the apex transmitted to the base. I wish you would all listen to it. It is not very distinct, but it is worth hearing.

The temperature is 98.7° F.

VISITOR: When did he have the hemorrhages?

DR. HAMBURGER: He had them before his admission. It is simply stated in the history. He originally came to the hospital with a history of illness of some eight months' duration. He

then complained of anorexia, loss of weight, dyspnea on moderate exertion, and two hemorrhages from the mouth.

The *lungs* are negative.

Eyes.—His pupils are equal. You see the pallor of the sclera and also a suggestion of the yellowish color.

Gums.—See the pus that can be expressed. All the teeth show about the same thing. He has rather a severe pyorrhea.

Throat.—His tonsils are enlarged and thickened, particularly the left one, but otherwise negative. The mucous membrane of the mouth is pale. There are several areas of pigmentation. You see on this right cheek two brown spots.

Tongue.—No pigmentations. The tongue is rather smooth and shiny and glistening, showing atrophy of the papillæ.

Neck is negative. He has no palpable thyroid; no cervical adenopathy; no tracheal tug. There are no glands palpable.

Abdomen.—A little bit distended in the lower portion. Practically free from tenderness.

When he was here in August he had a palpable spleen, but no sign of that can be found now. The liver is not palpable. To all intents and purposes the abdomen is negative.

Extremities show that slight yellowish-lemon tinge. Tibial crests are a bit rough, more marked on the left leg than the right. The extremities likewise show loss of weight and flabbiness of the tissues.

Knee-jerks are present, perhaps somewhat exaggerated on the right side. It is difficult to bring out the ankle-jerks. He has no edema anywhere. What have we here?

VISITOR: Pernicious anemia.

DR. HAMBURGER: Correct. Could it be anything else? How about the hematemesis? He has no lung symptoms or findings. He has no cough. Do you get hematemesis in pernicious anemia?

VISITOR: Yes.

DR. HAMBURGER: You do. You can. What is it from? You get bleeding in pernicious anemia either as cause or effect. The history of two hemorrhages does not rule out pernicious anemia. In a series of 670 cases of pernicious anemia collected

some years ago 10 showed a history of stomach bleeding; that is a small percentage. But it does occur, not only hemorrhage from the mouth, but hemorrhage from any part of the body.

Le Count and Hirsch have drawn attention to the frequency of petechial hemorrhages in the stomach of alcoholics and of patients dying from delirium tremens. Hirsch studied a number of these stomachs in patients dying from delirium tremens, and concluded that these petechial hemorrhages were not the result of hemorrhagic gastritis, but that they were localized hemorrhages caused by a toxemia. We have recently had on the service here a young man entering the hospital suffering from profound and repeated gastro-intestinal hemorrhage, in whom, following his fourth hemorrhage, developed an acute delirium of the nature of delirium tremens. He was irrational for nearly ninety-six hours, finally becoming normal, and made an uneventful recovery.

The source of the bleeding is probably the most interesting study. In addition to the bleeding from ulcer, hemorrhagic erosions, and dilated esophageal veins discussed here, hematemesis has been reported in disease of the gall-bladder and disease of the appendix. In connection with the latter, Outland and Clendening[1] have reported a case of hematemesis due to chronic appendicitis, the hemorrhage stopping only after a chronic appendix buried in dense adhesions had been removed. They call attention to the fact that Moynihan, La Roque, and others had described similar cases.

Donald C. Balfour[2] reports an interesting case of a recurring gastro-intestinal hemorrhage where gastric and duodenal ulcer, gall-bladder, varicose gastric veins, erosions of the gastric mucous membrane, cirrhosis of the liver were all excluded by several exploratory laparotomies, and only following the removal of the spleen were the hemorrhages stopped. Balfour comments on the difficulty of the correct interpretation of gastro-intestinal

[1] "Hematemesis Due to Chronic Appendicitis with an Explanation of Its Pathology and Physiology," Amer. Jour. Med. Sci., Feb., 1917.

[2] "Splenectomy for Repeated Gastro-intestinal Hemorrhages," Annals of Surgery, January, 1917.

hemorrhages, and calls attention to the intimate relationship of spleen and liver, the excellent results of splenectomy following splenic anemia, concluding that in many obscure cases the splenic factor should be considered.

Moschowitz[1] reports 4 interesting cases "upon whom the unequivocable diagnosis of bleeding ulcer of the stomach had been made both by myself and by several very competent colleagues, and yet all of the operations failed to reveal even the slightest tangible lesion of the stomach. All of these patients recovered."

Moschowitz concluded that his 4 cases are to be grouped under the *exulceratio simplex* of Dieulafoy.

Even of greater interest than these cases of hematemesis are the cases of fatal gastro-intestinal hemorrhage where autopsy fails to reveal any significant pathology whatsoever. Armstrong and Mullally[2] report a case of fatal gastro-intestinal hemorrhage where the autopsy findings were completely negative, and offer no explanation of the bleeding. A few weeks ago a patient was admitted to our service, apparently not seriously ill, and complaining only of slight cough and dyspnea. The examining room diagnosis was "bronchial asthma." He came into the ward about 8 o'clock in the evening, and about midnight the nurse, on making her night rounds, noticed he was bleeding from his nose and mouth. Before the intern could respond to the emergency summons the patient was dead from a fulminant mouth and nose hemorrhage.

Autopsy by Dr. Wells the following day failed to reveal any positive source of the bleeding, although the postmortem was done with the greatest possible care from the roof of the mouth, brain, and skull down to the extremities. With the exception of a slightly increased bile staining of the liver nothing abnormal was found. This may have been an early cirrhosis of the liver, with bleeding from the simultaneous rupture of many capillaries, similar to some of Preble's cases.

[1] "Massive Hemorrhages from the Stomach Without Demonstrable Ulcer," Amer. Jour. Med. Sci., November, 1916.

[2] "Fatal Gastro-intestinal Hemorrhage," Amer. Jour. Med. Sci., June, 1914.

CLINIC OF DRS. ISAAC A. ABT AND DAVID C. STRAUS

MICHAEL REESE HOSPITAL (SARAH MORRIS MEMORIAL HOSPITAL FOR CHILDREN)

SPONTANEOUS PNEUMOTHORAX DUE TO EMPHYSEMA

Report of a Case with Autopsy Findings in the Lungs. Technic of Valvular Drainage. Principal Types of Spontaneous Pneumothorax. Etiologic Factors.

March 3, 1917.

DR. ABT: This morning I desire to present a very interesting case that came in during the early part of the week, a child showing marked symptoms of pneumothorax. The case was such that we needed the aid of a surgeon. Dr. David C. Straus, of the surgical department, kindly helped us, remaining with the case until the unfortunate death of the child. I have asked him, on account of his knowledge and experience in this kind of work, to present its points to us—the pathology, the complications, the *x*-ray features, etc.

DR. STRAUS: The child, a boy twelve years of age, was brought to the hospital by his mother because of dyspnea, which was so severe that he could not lie down, but had to remain in an upright posture in order to breathe. This severe dyspnea began suddenly three days before the child was brought to the hospital. It had progressively become worse, so that when the child was first seen at the hospital he was in a condition of severe orthopnea and very cyanotic. The mother stated that the child had been apparently well up to about four weeks ago, when he contracted what she called a "cold." Previous to that time he seemed healthy, though, she said, he had always been a rather weak child and had been coughing somewhat for the past year. He had had no night-sweats, however, no loss of

appetite or weight, nor did he present any other symptoms suggesting tuberculosis of the lung. This, of course, is the first condition thought of when a child develops pneumothorax because from 76 to 90 per cent. of all cases of pneumothorax are secondary to tuberculosis of the lungs. The people were poor, and the child, compelled to work in a bakery practically the whole day, had not been allowed to spend much time out of doors.

He entered the hospital on the service of Dr. Abt, and I would like to have him tell you the condition in which he found the child on entrance.

Dr. Abt: The boy came in looking pale, moderately cyanotic, and terrifically dyspneic. His condition was so desperate that he was unable to lie in bed, and his suffering seemed so intense that we made but a hurried examination. On percussion we found a condition approximating tense tympany on the right side. We felt that there was some pathology in the right lung. On the left side the conditions seemed normal, though on auscultation and percussion a mild emphysema suggested itself. There being no abdominal distention nor any distention of the stomach, we thought that there was some condition the nature of which we were not positive, possibly empyema or some marked enlargement in the mediastinum causing pressure. The dyspnea was so very severe that the child was simply wearing himself out in his efforts to breathe. The condition of the patient was so pathetic that we thought we would make no more extended examination, and hurried him off to the *x*-ray room to confirm our suspicions in the case. The report came back at once that the right pleural cavity was filled with air and the right lung was collapsed.

Dr. Straus: I saw the child about noon, through the courtesy of Dr. Jampolis, who told me that the röntgenologic examination had shown that the child had a right-sided pneumothorax, and that Dr. Abt had instructed him to insert a needle into the right pleural cavity to allow the air free in the pleural cavity to escape. Dr. Jampolis inserted the needle in the seventh intercostal space in the posterior axillary line. At

once one could hear an escape of air, immediately after which the child's condition improved perceptibly. A small rubber tube was attached to the needle, and at each expiration the air was allowed to escape from the pleural cavity, while at each inspiration the air was prevented from entering the thorax through the tube. To maintain this arrangement a piece of soft dental rubber-dam was so placed over the free end of the rubber tube that air could escape from the chest, but none could enter from without. The needle, with this contrivance attached, was allowed to remain in the chest until evening. At this time the child's condition had gradually grown worse again, his pulse having risen to 140, his respirations to 48, and I was asked to see him.

He was in an extremely precarious condition when I saw him, and it seemed that we would lose him unless something radical was done at once. There was a considerable amount of subcutaneous emphysema beneath the skin about the needle and all about this side of the chest. It even extended up on the neck and face, the right side of which was considerably puffed up, resembling somewhat a case of parotitis. A hasty physical examination at this time showed that the left lung was emphysematous to such a degree that it obliterated the cardiac dulness. This was believed at the time to be a compensatory emphysema. I at once inserted a much larger needle, to which I connected a Potain aspirating bottle. I had one of the house staff exhaust the air from the bottle. When a good vacuum was formed within, the valve was opened on the suction arm, which was connected with the needle in the chest. One could then hear the rush of escaping air. This aspiration was repeated several times. The child's condition, under this treatment, improved markedly. The cyanosis decreased perceptibly, and his pulse became slower and better in quality. As soon, however, as the aspiration was discontinued for a few minutes the child's condition grew worse again. As this pumping could not be kept up indefinitely, it was clear that we would have to resort to more radical measures. An immediate operation to remove the pressure seemed imperative.

Consequently, while the aspiration was being continued, the area about the needle was prepared for operation and anesthetized locally with ½ per cent. novocain solution. Then the needle was withdrawn, and an incision about 2 cm. long was made through the skin, along the upper border of the eighth rib, so as to avoid the intercostal vessels which run along the lower margin of the rib above. The skin edges being held retracted, a small stab incision was made through the intercostal muscles. This opening was enlarged by inserting an artery forceps with the jaws closed and then opening the jaws. In this way the pleural cavity was entered without causing any bleeding. As soon as the pleura was opened there was a rush of air that reminded one of a puncture of an automobile tire. A piece of gauze was at once held over the opening to prevent the too rapid escape of air. It is dangerous to relieve the pressure too suddenly because it may cause a sudden change in the position of the heart and great vessels. When the rush of air had subsided the respiration became slower and a great deal easier. The child, who had hitherto been unable to breathe except in an upright posture, was now able to lie down. To maintain the opening a metal tracheotomy tube was inserted, and the muscles and skin closed about this by means of a few interrupted stitches. The tube was held in place by means of wide strips of adhesive plaster.

If the simple reduction of pressure fails to give sufficient relief, as was the case with our patient, all authorities agree that it is necessary to establish an opening between the pleural cavity and the outside air.

Some authorities, as Bandelier and Roepke, "Die Klinik der Tuberkulose," 1914, p. 347, believe that in a case such as this one (valvular pneumothorax) a large *free* communication should be maintained between the pleural cavity and the outside, so that the lung will remain collapsed. They recommend the Unverricht procedure which consists in making a long incision in an intercostal space, inserting the largest possible drainage-tube, and then merely covering the end of the tube with a piece of thin gauze so that the *free ingress and egress* of air can

take place. In these cases they condemn valvular drainage, which consists of draining by means of a tube so arranged that it will allow the air in the pleural cavity to escape from the chest, but will not permit the outside air to enter the thorax through this drainage-tube. Valvular drainage was first instituted by v. Bramann, in 1893, and can be easily effected by a device suggested by Thiersch. This consists in slipping a finger-cot just over the end of the rubber drainage-tube, and tying a ligature about it so that it will remain in place, and cutting away the closed end of the finger-cot. The thin rubber appendix to the drainage-tube, thus formed, will allow the air to escape from the chest, but during inspiration will collapse and so prevent air from re-entering. There are other methods of accomplishing valvular drainage, as by means of the Tiegel metal drain, which is built for this purpose, or by means of the arrangement I used, which I shall tell you about later. Bandelier and Roepke do not believe that valvular drainage should be instituted in any form of pneumothorax, for they believe that this tends to prevent the spontaneous closure of the opening in the lung, or may even reopen it in case the opening has become sealed.

Other authorities, however, as Garrè ("Lungenchirurgie," Garrè and Quincke, 1912, p. 80) and Sauerbruch ("Handbuch der praktischen Chirurgie," 1913, vol. ii, p. 790), *recommend the use of valvular drainage in valvular pneumothorax.*

At first, following the advice of Bandelier and Roepke, I left the opening in the metal tracheotomy tube uncovered, so that the air passed freely to and from the pleural cavity. However, the child's breathing under the circumstances was not satisfactory.

It seemed essential to maintain negative pressure in the right pleural cavity, so that the right lung could perform its physiologic function, as far as this could be maintained, for it was clear that the child was unable to breathe satisfactorily with the use of the left lung only. Just why this was so was not clear at that time, but the explanation was evident later and will be referred to again. Moreover, valvular drainage tends to prevent the mediastinum from flapping from the right to the left side of the

chest during each respiration, which of itself is sufficient to cause the patient distressing and even dangerous respiratory and cardiac symptoms. I therefore decided to employ valvular drainage. To accomplish this I placed a large square piece of rubber dental dam over the external opening of the tracheotomy tube, and held this in place by means of strips of adhesive plaster, so placed about the margins of the piece of rubber that air could escape from the chest during inspiration by displacing the rubber at the end of the tube; whereas, during expiration, air was prevented from entering the chest through the tube, for the rubber was then drawn up against the opening due to the negative pressure within. While negative pressure was maintained in the right pleural cavity in this way the right lung could expand somewhat.

This arrangement proving to be more satisfactory than leaving the tube wide open, was maintained in place and covered with a light gauze dressing. The child was then returned to bed and laid on the affected side so that he would have full excursion of the left lung. He was comparatively comfortable now and slept at intervals. His mother said that this sleep was the first he had had in three days. His respirations were still labored, however, and he slept only for short periods at a time. At 4 A. M., five hours after the operation, the pulse was 140 and the respirations 46. At 8 A. M. the pulse was 140 and the respirations 36. During the previous day the child had been given paregoric in 20-drop doses every four hours. In addition to this he was given, after the operation, morphin sulphate hypodermically. This was repeated as indicated. The day following the operation the temperature varied between 98.8° and 100.6° F.; the pulse came down to as low as 120 and the respirations as low as 36. The subcutaneous emphysema spread slightly after the operation, but was never of high grade. Subcutaneous emphysema is dangerous only when it is of extremely high grade and mechanically interferes with respiration.

Before proceeding with the further course of this case I would like to say a few words about spontaneous pneumothorax cases in general. As regards etiology, *pulmonary tuberculosis*

is by far the commonest anatomic cause of pneumothorax. It is the etiologic factor in 86 per cent. of all cases according to Rose; in 90 per cent. according to Gerhardt. Biach ("Wien. med. Wochenschr.," 1880, xxx) collected all the cases of pneumothorax from the records of the three great hospitals in Vienna during a period of thirty-eight years. There were 918 cases of pneumothorax with the following etiology:

Tuberculosis of the lung	715
Gangrene of the lung	65
Empyema	45
Injury	32
Bronchiectasis	10
Abscess of lung	10
Emphysema	7
Breaking down of a hemorrhagic infarct	4
Paracentesis thoracis	3
Perforation of esophagus	2
Perforation of stomach	2
Nematode worms in pleural cavity	2
Rupture of an encapsulated peritoneal effusion	1
Hydatid of lung	1
Caries of ribs	1
Rupture of bronchial glands	1
Abscess of breast	1
Fistula between pleura and colon resulting from hydatids	1
Uncertain causes	14

Without going further into the etiology and pathogenesis of pneumothorax, it might be well to mention that there are three principal types of spontaneous pneumothorax. These are: *closed pneumothorax*, *open pneumothorax*, and *valvular pneumothorax*. The case we are considering is of the last variety.

By a *closed pneumothorax* we mean that the tear in the lung has completely closed. If the opening or tear in the lung is small the air escapes more or less gradually, and, therefore, the symptoms are not at all violent. Indeed, we do not know how frequent this form really is, for in cases in which the symptoms are very slight or are entirely absent it doubtless occurs often without being diagnosed. The essential feature of this type is the fact that when the pressure of the escaped air reaches a certain degree the lung becomes compressed and the rent in the

lung is sealed; that is, the perforation becomes completely closed. Following this the escaped air in the pleural cavity is gradually absorbed, and then the lung expands again. The only treatment indicated in closed pneumothorax with slight symptoms is the initial administration of morphin to combat the acute symptoms, and the application of compression to the affected side of the chest by means of strapping it with adhesive plaster, and having the patient lie on the affected side. This not only puts the diseased lung at rest, but also gives the healthy lung the maximum opportunity to expand, so that it is best able to take on the additional work thrown upon it. Where the symptoms are marked the case is treated by the use of a needle or aspirating the contained air, as was done in this case.

Open pneumothorax is the name given to the pneumothorax where the point of perforation remains open, so that the air on respiration constantly passes in and out of the pleural cavity. This type is not as urgent as the third or valvular type, for the pressure of the escaped air never becomes positive, but only can become the same as the atmospheric pressure.

The third, and clinically the *most frequent and most dangerous type*, is *valvular pneumothorax*. In this form air enters the pleural cavity at each inspiration, but on expiration a valve-like closure of the perforation prevents the air free in the pleural cavity from escaping. Thus with each inspiration air accumulates in the pleural cavity that cannot escape again. Later, if the pressure in the pleural cavity increases so that no more air can enter it on inspiration, the valvular pneumothorax may become closed. The great danger in cases of pneumothorax is due to the displacement of important structures, chiefly the mediastinum, heart, and the great vessels. Displacement of the mediastinum compresses the opposite lung and so reduces the only available functionating lung tissue, so that the patient's respiration becomes more and more difficult and insufficient, while displacement of the heart and large vessels if marked, by interfering with the circulation, may cause sudden death.

It is often impossible to make a clinical diagnosis of the type of pneumothorax in a given case, but this is not of particular

importance because the treatment depends on the urgency of the symptoms, and should be systematically carried out as indicated above. In case an open pneumothorax shows no tendency to close, an open operation may later be considered for the purpose of closing the opening by suturing the lung. A person may, however, go on for months or even years with an open pneumothorax.

In the case we are considering today we are dealing with a case of valvular pneumothorax in which the symptoms caused by positive pressure in his chest made it imperative to perform an open operation in order to relieve this pressure. The röntgenograph (Fig. 215) shows how the heart and mediastinum were pushed and displaced to the left, and that the right lung was collapsed and lay against the hilus. The picture also shows a few fine adhesions extending from the apex of the right lung to the parietal pleura at the apex of the pleural cavity. It is to be observed, also, that the lung is bound down to the diaphragm by adhesions between the visceral pleura of the lower lobe and the diaphragm. The right lung, even though collapsed, is seen to be emphysematous, and shows no sign of any shadow near the periphery that would lead one to suspect a tuberculous process. The left lung presents the typical picture of emphysema of quite high grade, and shows some peribronchial infiltration. No enlarged glands are to be seen at the hilus on either side.

No röntgenologic examination was made after the tracheotomy tube was inserted.

The treatment already described was kept up for three and a half days. His condition gradually grew worse, and at the end of this time the child died.

Although permission could not be obtained to perform a complete necropsy, we were permitted to remove the lungs. This we did by enlarging the intercostal incision made to introduce the tracheotomy tube.

On opening the right side of the chest, the hand was introduced and the right lung and pleural cavity palpated and then inspected. The parietal and visceral pleuræ were everywhere

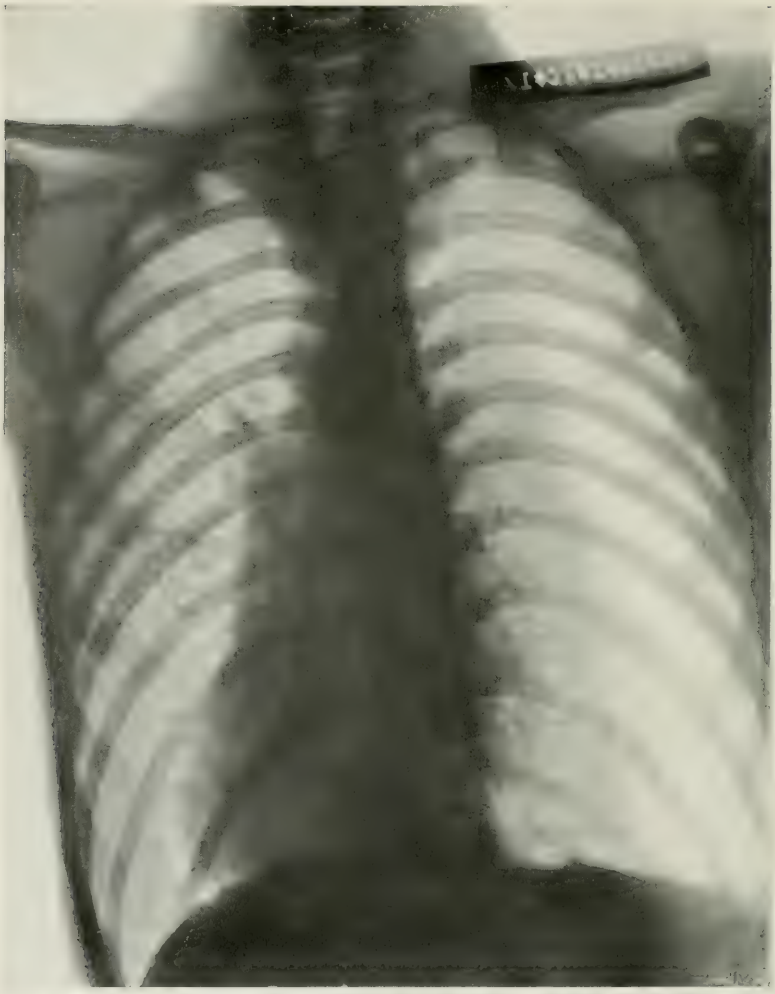


Fig. 215.—Röntgenograph of the case of valvular pneumothorax due to emphysema, taken when the child entered the hospital, before a needle was introduced into the right pleural cavity. Plate reversed. Note that the right pleural cavity is filled with air, that the right lung is collapsed but is emphysematous, and that there are a few adhesions at the apex of the right lung, and that the base of the right lower lobe is adherent to the diaphragm. Also note that the mediastinum is displaced to the left, as is also the heart. The left lung is seen to be emphysematous, and peribronchial infiltration is plainly to be made out.

smooth and glistening. The parietal pleura appeared entirely normal as far as it could be inspected. There was no fluid in

the pleural cavity, no serous effusion, no blood, and no pus. This was quite in accord with whatever evidence we had as to the cause of the perforation, for, as stated before, neither the history nor the röntgenogram indicated tuberculosis, gangrene of the lung, or empyema that had ruptured, which, as was stated before, are the usual etiologic conditions in order of their frequency. Nor did the clinical picture suggest any of these conditions. It was clear, therefore, from the absence of exudate that the air had escaped through a non-infected opening.

Fig. 216.—Photograph of the lungs immediately after removal. Note the bullous emphysema in both lungs.

Palpation showed the presence of the few adhesions at the apex and at the base that can be seen in the röntgenograph (Fig. 215). The right lung was now cut away at the hilus and removed. Next the mediastinum was palpated and was found to be slightly emphysematous. The mediastinum was now torn through and the left lung removed. There were no adhesions on the left side of the thorax. The lung was free and was readily removed. The left pleura seemed negative on palpation. It could not be inspected.

The two lungs resembled one another closely. This can be seen from a photograph that was taken very soon after they

were removed (Fig. 216). Both lungs contained air and floated on water. The surface was very roughly nodular, due to large

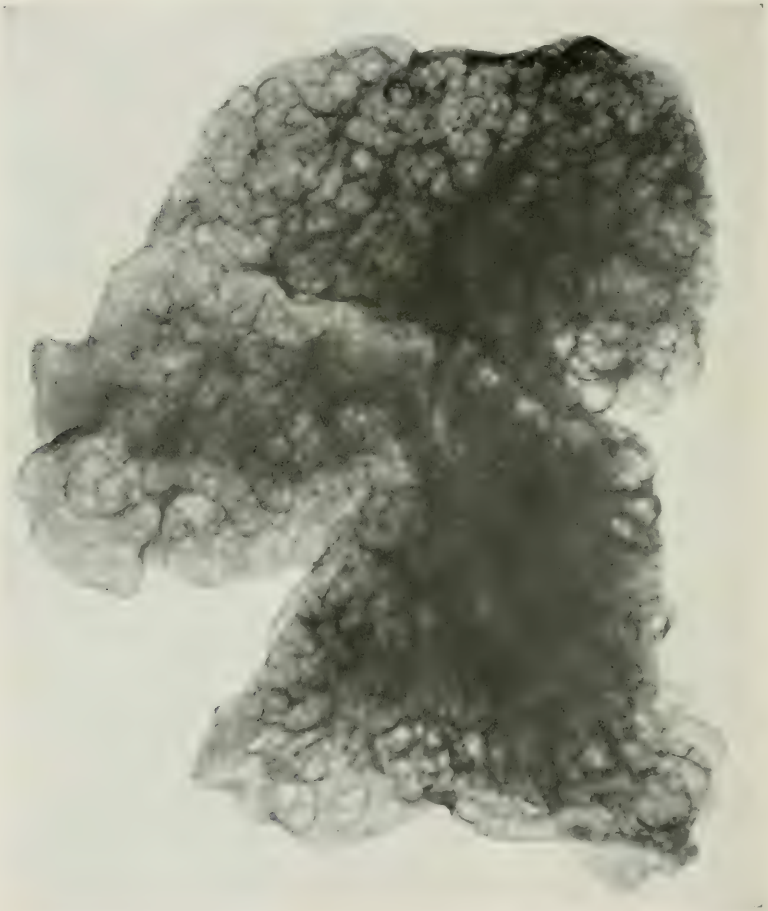


Fig. 217.—Röntgenograph of the right lung taken immediately after removal. This shows that the essential lesion present is bullous emphysema. No suggestion of tuberculosis whatsoever at the periphery of the lung. The site of perforation does not show in the picture, but this was found to be definitely due to the rupture of one of the bullæ, and this was located at the lower portion of the right upper lobe, posteriorly.

bullous emphysematous blebs. The very high grade of emphysema can best be appreciated by looking at the radiographs of

the lungs (Figs. 217, 218) which I had taken almost immediately after they were removed before the air had time to become absorbed. The specimens which I shall pass about

Fig. 218.—Röntgenograph of the left lung taken immediately after removal. The conditions here are seen to be practically identical with that found in the right lung.

give only a very poor idea of the original picture, which is seen to the greatest advantage in the photograph and the röntgenographs.

You will be able, however, to see the site of perforation in

the right lung. The broom-straw is inserted in the perforated area. This you will see is in the right upper lobe, posteriorly, rather low down, almost at the fissure. You will note that the perforation is round and of rather fair size. At the time of autopsy it appeared to be about the size of a "B" shot. Of course when the lung was distended it must have been considerably larger. It lies very definitely at the center of the periphery of a very large emphysematous bulla. The röntgenographs show that the lungs are rather normal except for the bullous emphysema. On section, however, both lungs showed some small areas of peribronchitis and bronchitis, and there were some areas of bronchopneumonia in the lower portion of the left upper lobe and in the upper portion of the left lower lobe. The röntgenogram shows peribronchial infiltration in this portion of the left lung, as you can see. Both lungs showed some slight bronchiectasis and a small amount of interstitial emphysema. The slight interstitial emphysema in the lungs as well as that in the mediastinum probably spread from the site of perforation in the lung, and probably was of no clinical importance as it was so slight.

These findings were all in accord with the history and the clinical findings, which I shall refer to in a moment.

The prognosis in valvular pneumothorax is always grave even when properly treated. In pneumothorax due to tuberculosis the mortality is very high, about 80 per cent., though instances have been known where spontaneous cure occurred. In our case the boy had no chance of recovery. This is clear now, for he was unable to get along with the left lung alone, as this was so emphysematous.

Taking all the evidence together, the explanation of the case seems to me to about as follows:

The boy, who was in the bakery daily, where he was exposed to the alternate heat from the ovens and to cold, probably developed a chronic bronchitis. This was perhaps the cause of the slight cough that the mother stated the boy had had for the past year or so.

In this connection it may be interesting to know that of the

occupational diseases of bakers, diseases of the respiratory tract occupy first place. Hirt's German statistics (quoted by Kober and Hanson, "Diseases of Occupation and Vocational Hygiene," 1916, p. 687) show that in bakers 28 per cent. of all deaths from internal diseases are due to diseases of the respiratory tract, 7 per cent. being due to pulmonary tuberculosis, 8.4 per cent. to pneumonia, and that emphysema, chronic bronchitis, and asthma (miller's asthma) are common.

Hoffman's statistics (Bulletin, Bureau of Labor, No. 82, May 1909, p. 534), based on 1357 deaths from all causes among bakers in the United States, show a death-rate of 34 per cent. from diseases of the lungs and air-passages.

Weyl ("Handbuch der Arbeiterkrankheiten," 1908, pp. 487–490) states that among 183 bakers treated for internal diseases in the Munich hospitals during the year 1889, there were

Lung and chest diseases	60
Infectious diseases	54
Stomach and intestinal catarrh	23
Rheumatism and gout	20
Heart diseases	8
Kidney diseases	6
Angina	6
Marasmus	5
Malaria	1

He further states that of 1000 members of the Vienna bakers' guild, from 1890 to 1893, there were yearly an average of 102 who were incapacitated from acute or chronic diseases of the respiratory organs. Of these 102 cases, 58 suffered from acute or chronic bronchial catarrh, 5 from emphysema, 6 from pneumonia, and 5 from pleurisy. Diseases of the respiratory tract were the cause of 33.8 per cent. of the illness.

Of the diseases of the deeper air-passages in persons who work in dusty and bad air, chronic bronchitis is the commonest. Although flour dust is classed with the indifferent kinds of dust, nevertheless, if inhaled in large amounts it leads to damage of the bronchi. The flour dust, which is made up of round, small particles of flour, which are not sharp and do not traumatize, is mixed with the saliva in the mouth and the bronchial mucus

in the deeper air-passages, and forms a sticky paste which is brought up and expectorated after severe coughing ("bakers' cough").

In the course of time the smallest bronchi become plugged up, and in addition to atelectasis of the corresponding alveoli, *emphysema* develops in the neighboring parts of the lung.

The boy, as a result of inhaling flour frequently, may thus have developed the slight amount of bronchiectasis which we found. At all events, it seems probable that the "cold" from which he suffered for four weeks just before he entered the hospital may have been due to, or may have been the cause of, the moderate bronchopneumonia that was discovered post-mortem, which was evidently the cause of the slight fever that he ran the entire time he was in the hospital. It seems possible that during this month of sickness, due to unusual coughing, he developed an increase in his emphysema, and perhaps it then developed the high grade found at necropsy. That this was of subacute nature was shown by the microscopic sections, which show that the walls of the large bullæ are rather thick and are infiltrated with round cells, and, therefore, could not have been terminal, but must have been, at least, subacute.

Apparently during a coughing spell one of the large bullæ ruptured spontaneously. This led to the acute valvular pneumothorax, which was hopeless, because the remaining or left lung was so diseased that it was inadequate to carry on respiration satisfactorily, and put a severe strain on the heart which wore the child out. The case was, therefore, apparently hopeless from the outset.

The case is certainly most unusual. How rare the condition really is can best be appreciated by referring to the Vienna statistics quoted at the outset, which, you may remember, show that in Vienna, in thirty-eight years, there were only 7 cases of spontaneous pneumothorax due to emphysema.

CLINIC OF DR. FRANK WRIGHT

NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

ACUTE NEPHRITIS FOLLOWING TONSILLITIS

Rapid Reduction of Edema. Early Tonsillectomy. Estimation of Kidney Function.

THAT acute nephritis may follow a sore throat is a matter of frequent record—the nephritis may vary from a simple affair, with scanty febrile urine containing traces of albumin and a few casts, to one of much greater degree. This patient is shown to you today because he is one of four seen since the first of the year showing acute nephritis of marked degree with no evidence of origin other than infection in the throat.

On New Year's Eve this young man first felt a soreness in his throat, with the usual evidences of infection accompanying it. He took no medicine, but went to bed, remaining there the following day; on the third day called a physician, who promptly recognized a tonsillitis with peritonsillar infection, and twenty-four hours later evacuated considerable pus from the right tonsil by free incision. This afforded great relief. Little medication was prescribed other than cathartics, and the boy remained in his room until January 10th, after which he resumed his work as machinists' helper, although feeling somewhat weak and lazy, the throat remaining "raw." Upon awakening January 23d he found his face noticeably swollen, especially about the eyes, but this disappeared about 9 o'clock. The two following days the same swelling was found, requiring somewhat longer to disappear each morning. At this time no other symptoms were noted. January 26th the feet were also swollen over the

insteps, and for the first time the patient did not go to work. In spite of the day's rest, the edema was more marked; the following morning he was unable to put on his shoes, and that afternoon his attention was attracted to his urine by the fact of its being scant, feeling hot when passed, and being distinctly bloody.

Again he called the physician, who told him of the nephritis, directed that he maintain a non-nitrogenous diet, in addition ordered cream-of-tartar lemonade, and very properly suggested that he leave his boarding-house for a hospital. This the patient did not do, but waited two days longer, during which time the urine became more scanty and more bloody, 10 and 12 ounces being passed in twenty-four hours.

Marked dyspnea and palpitation on slight exertion appeared on the 29th, and the patient then journeyed from Cincinnati to Chicago, being obliged to sit up the entire night on the train. At the station curiosity caused him to ascertain his weight, and the scale recorded a gain of 22 pounds over his weight as last taken during health. Upon entering Michael Reese Hospital there were noted the following salient points:

1. Marked edema of the whole face and especially the eyelids.

2. Deeply coated tongue, breath foul, tonsils injected, soft palate and pillars edematous, pharynx deeply injected.

The thorax moved freely. There were no signs of free fluid. The area of precordial dulness was slightly enlarged, the apex was in the fifth intercostal space almost beneath the nipple, and the aortic second sound was accentuated.

The abdominal muscles were soft and relaxed, no organs palpable, but on putting the patient in the knee-elbow position an area of dulness was elicited on percussing from below. This was taken as an evidence of free fluid, especially as it disappeared soon after improvement took place.

Edema of the genitalia was slight, but of the lower extremities very marked, especially below the knees. Temperature was 98.6° F., pulse 84, blood-pressure 210 systolic, 120 diastolic. Blood examination showed hemoglobin 80 per cent., red blood-

cells 4,400,000, white blood-cells 19,300. A single specimen of urine was acid, 1016 specific gravity, bloody, containing albumin, sediment packed with red blood-cells and 6 to 8 white blood-cells to high-power field. Cultures from pus expressed from the tonsil gave diplococci in pairs and chains, some encapsulated; staphylococci also present.

Behavior during the first six days in the hospital:

Day.	Diet.	Urine, c.c.	Weight, pounds	Blood-pressure.	Electric light pack.
1	Milk, 960 c.c.; no other fluid.....	980	151	210–120	Given
2	Milk, 1200 c.c.; water, 1200 c.c.....	1500	144	180–105	"
3	Milk, 1000 c.c.; water, 1000 c.c.....	3250	138	145–100	"
4	Milk, 1000 c.c.; water, 1000 c.c.....	2220	131½	145–100	"
5	Milk, 1000 c.c.; water, 1000 c.c. Bread, butter, and fruit.	1020	127	125– 90	"
6	Milk, 1000 c.c.; water, 1000 c.c. Bread, butter, and stewed fruit.	510	125¾	120– 80	"

The urine no longer appeared bloody macroscopically, but the amount of albumin increased from a small amount to a distinct finding. While the urine was clear and light yellow, many shadow corpuscles were found, white corpuscles persisted, and hyaline and granular casts, which at first I was unable to find, became numerous.

The behavior during the first six days' stay was marked by a loss of 25 pounds in body weight and a reduction of blood-pressure from 210 to 120, and this by using milk and water combined with the electric light pack. No medication was used except 5 grains of blue mass on the day of entrance followed by 1 ounce of magnesium sulphate the following evening.

On the fifth and sixth days a small amount of bread and butter with stewed fruit was added, and, as I have so often found, this markedly interfered with the diuretic action of the milk.

At this time a Mosenthal diet was given for two days. The results of the second day are given.

Table I

Hours	Quantity Urine	Sp. gr.	Sodium Per Cent.	Sodium Quantity	Chloride Per Cent.	Chloride Quantity	Nitrogen Per Cent	Nitrogen Quantity
8-10 A.M.	182	1007	.235 %		.64 %			
10-12 Noon	122	1010	.52		.63			
12-2 P.M.	90	1018	.61		.55			
2-4 "	131	1020	.66		.85			
4-6 "	110	1017	.49		.54			
6-8 "	71	1016	.96		.68			
Total Day	706				3.89		.65	455
Night (8 P.M.-8 A.M.)	680	1015	.59		3.98		.82	557
Total 24 hours	1386	Maximum variation 10			7.87			1012
Intake	1800				10.			1260
	+414				+2.13			+248

Chart I

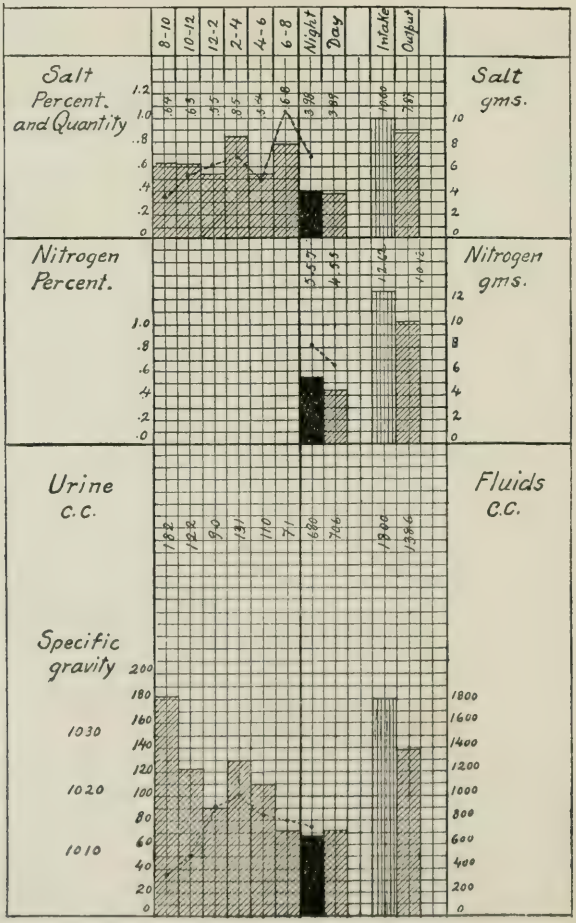
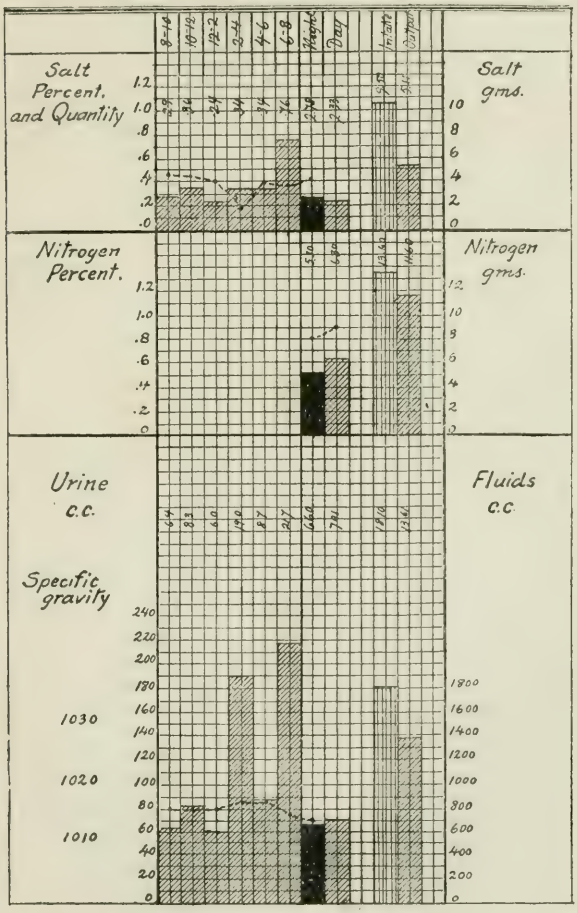


Table II

Hours	Quantity Urine	Sp. gr.	Sodium Per Cent.	Chloride Quantity	Nitrogen Per Cent	Nitrogen Quantity
8-10 A.M.	64	1016	.46 %	.29 gm		
10-12 Noon	83	1016	.43	36		
12-2 P.M.	60	1016	.40	24		
2-4 "	190	1017	.18	34		
4-6 "	87	1017	.39	34		
6-8 "	247	1015	.35	76		
Total Day	701			233	.90 %	6.30
Night (8 P.M.-8 A.M.)	660	1014	.42	278	.81	5.30
Total 24 hours	1361	Maximum 1017 Minimum 1014		511		11.60
Intake	1810			950		13.50
	+449			+439		+1.9

Chart II



The points of interest are the increase in the amount of night urine and the passing of more salt and nitrogen at night than during the day. Fixation of gravity was not present at this time. There was evidence of renal fatigue in the evening.

The kidney is able to concentrate salt and nitrogen, as shown by the approach to 1 per cent. in each case. Mere traces of albumin were present, but casts were numerous and shadow corpuscles + white blood-cells were still present in large numbers.

For five days a non-nitrogenous salt-free diet was then maintained, the urine passing freely and the weight being maintained. A constant soreness of the throat was complained of, however, and two weeks after entrance the tonsils were removed under local anesthesia by Dr. Harry Kahn. Considerable pus could be expressed from the right tonsil. No ill effect upon the kidney followed the early removal. The volume of urine was lessened, but this was undoubtedly due to the restriction in the intake of fluid.

Four days later the non-protein salt-free diet was resumed, the urine giving a very faint trace of albumin, hyaline, and granular casts. Very few shadow red corpuscles were found, but white blood-corpuscles were present in numbers exceeding the red cells.

On the twentieth and twenty-first days of his stay in the hospital the Mosenthal diet was repeated. The results of the second day are given that you may compare them with those obtained two weeks previously. These results show how constantly the kidney works when under the same conditions, and in this case how the elasticity is interfered with as shown by the fixation of specific gravity.

In the first test, however, the night excretion of both salt and nitrogen was greater than the day. The second trial shows that the nitrogen elimination is improving earlier than the salt, but there is still a great excess of fluid, salt, and nitrogen left over for a tardy excretion during the night.

In this case careful chemical examination of the blood failed to be of any material help as compared with the test for renal function.

INDEX TO VOLUME 2

ABDOMEN, characteristic, in splanchnoptosis, *Jan.*, 633
Abdominal aneurysm, *Jan.*, 673. See also *Aneurysm, abdominal.*
  belt in chronic constipation, *March*, 1090
  diseases, gangrene of lung from, *Nov.*, 563
  viscera, so-called ligaments of, *Jan.*, 639
    supporting structures, *Jan.*, 639
Abortive form of infantile paralysis, *Nov.*, 480
Abscess, blind, of teeth, relation of, to systemic diseases, *March*, 992
    roentgen-ray diagnosis, *March*, 995
  of prostate in gonorrheal arthritis, *May*, 1191
  pulmonary, after croupous pneumonia, *Jan.*, 661. See also *Pulmonary abscess.*
Achylia, chemical, *Jan.*, 700
  constitutional, *Jan.*, 699
  essential, *Jan.*, 699
  gastrica, *Jan.*, 691
    clinical history, *Jan.*, 698
    conditions simulating, Riegel's classification, *Jan.*, 704
    definition, *Jan.*, 696
    diagnosis, *Jan.*, 693, 695
      differential, conditions to consider in, *Jan.*, 704
      test-meal in, *Jan.*, 702
    epigastric pain in, *Jan.*, 698
    gastric motility in, *Jan.*, 700
    gastrogenic diarrhea in, *Jan.*, 702
    gastroptosis in, *Jan.*, 702
    historical notes on, *Jan.*, 696
    histories of cases, *Jan.*, 692, 694, 696, 704
    hunger-pains in, *Jan.*, 698
    physical examination, *Jan.*, 692, 695
    roentgen-ray examination, *Jan.*, 693, 695
    stomach emptying time in, *Jan.*, 701
    symptomatology, *Jan.*, 698
Achylia gastrica, treatment, *Jan.*, 693, 703
      dietetic, *Jan.*, 703
      medical, *Jan.*, 703
      psychic, *Jan.*, 704
      rest in, *Jan.*, 703
    visceroptosis in, *Jan.*, 702
  psychic, *Jan.*, 700
Acidosis, *Jan.*, 681
  factors in production of, *Jan.*, 682
  Fridericia method of estimating, *Jan.*, 683
  history of case, *Jan.*, 688
  in decomposition in infants, treatment, *Jan.*, 817
  in diabetes mellitus, *July*, 29
      decreasing fats in diet, *July*, 35
      dietetic treatment, *July*, 35, 51
      further report on, *July*, 36
      general considerations, *July*, 33
      history of case, *July*, 29
      laboratory findings, *July*, 31
      medicinal treatment, *July*, 35
      physical examination in, *July*, 30
      temperature in, *July*, 30
  Marriott's method of determining reserve alkalinity in, *Jan.*, 684
  methods of estimation applicable to blood, *Jan.*, 684
      difficulties in, *Jan.*, 685
    to urine, *Jan.*, 687
  newer methods of estimating, *Jan.*, 682
  subsequent history, *Jan.*, 689
  treatment, *Jan.*, 690
  Van Slyke's method of estimating reserve alkalinity in, *Jan.*, 686
Acne, *Sept.*, 317
  bacteriology, *Sept.*, 320
  cachecticorum, *Sept.*, 319
  dietary in etiology, *Sept.*, 321
  disturbances of sexual organs in etiology, *Sept.*, 320
  etiology, *Sept.*, 319
  indurata, *Sept.*, 318
  iodid, *Sept.*, 319
  necrotica, *Sept.*, 319
  puberty in etiology, *Sept.*, 321

Acne punctata, *Sept.*, 317
pustular, *Sept.*, 318
rosacea, *Sept.*, 318
scrofulosorum, *Sept.*, 319
treatment, by dermal curette, *Sept.*, 323
by facial massage, *Sept.*, 323
by roentgen rays, *Sept.*, 324
hygienic, *Sept.*, 323
local, *Sept.*, 323
proper mastication of food in, *Sept.*, 322
regulation of bowels in, *Sept.*, 322
removal of comedones in, *Sept.*, 323
systemic, *Sept.*, 322
vaccine, *Sept.*, 322
with face powders, *Sept.*, 325
with pastes, *Sept.*, 324
varioliformis, *Sept.*, 319
vulgaris, *Sept.*, 318
Actinotherapy in retroperitoneal sarcoma, *March*, 1054
Addison's disease, cardinal symptoms, *Nov.*, 551
causes, *Nov.*, 554
pathogenesis, *Nov.*, 553
skin pigmentation in, *Nov.*, 549, 552
treatment, *Nov.*, 556
syndrome, case presenting, *Nov.*, 547
diagnosis, *Nov.*, 555
history, *Nov.*, 547
melanoderma in, *Nov.*, 549
physical examination, *Nov.*, 548
prognosis, *Nov.*, 555
skin pigmentation in, *Nov.*, 549
treatment, *Nov.*, 556
Adrenalin, intraspinal injection of, in infantile paralysis, *Nov.*, 486
Agalactia, *July*, 123
Air bubble in fluoroscopy of stomach, *July*, 209
Albumin milk in artificial feeding, *Sept.*, 267
in decomposition in infants, *Jan.*, 815
Aldehyd reaction of Ehrlich for urobilinogen, *May*, 1094
Alimentary glycosuria, *Sept.*, 340
Alkali therapy in diabetes mellitus, *July*, 51
Alkalipenia in infants, *Jan.*, 800
Allen treatment of diabetes mellitus in young, *Sept.*, 328, 334
inanition in, *March*, 999
keeping urine sugar free in, *March*, 1005
limitations, *March*, 1004
origin and development of, *March*, 1001
Allen treatment of diabetes mellitus, period of observation before, *March*, 1004
raising carbohydrate tolerance in, *March*, 1006
rendering urine sugar free in, *March*, 1004
results from, *March*, 1003
starvation period, *March*, 1002, 1003
strenuous exercise in, *March*, 1003
technic, *March*, 1002
Alveolitis, etiology, *March*, 993
relation of, to systemic disease, *March*, 994
Amebic dysentery, *Jan.*, 679
diagnosis, *Jan.*, 680
history of case, *Jan.*, 679
physical examination, *Jan.*, 679
treatment, *Jan.*, 680
Amoss and Flexner's immune serum in infantile paralysis, *Nov.*, 484
Anal fissure from chronic constipation, *March*, 1080
Anaphylaxis, *March*, 863
active, *March*, 864
alimentary type, *March*, 865
definition, *March*, 863
in fish poisoning, *March*, 865
blood-examination, *March*, 867
diagnosis, *March*, 867
etiology, *March*, 868
history of case, *March*, 865
laboratory findings, *March*, 867
physical examination, *March*, 866
in rheumatoid arthritis, *March*, 869
blood-examination, *March*, 870
diagnosis, *March*, 870
history of case, *March*, 869
laboratory findings, *March*, 870
physical examination, *March*, 869
roentgen-ray examination, *March*, 869
in typhoid fever, *March*, 871
histories of cases, *March*, 871, 873, 877, 879, 885, 887, 889, 891
production of, *March*, 864
parenteral digestion in, *March*, 863
therapeutic, *March*, 868, 871
Anemia, aplastic, *May*, 1180
aregenerative, *May*, 1180
digitalis in, *July*, 11
hemolytic, from Bothriocephalus latus, *May*, 1179
relation of syphilis to, *May*, 1185
in protozoic enterocolitis, *May*, 1127
myelopathic, *May*, 1179
pernicious, *Sept.*, 395; *May*, 1175. See also *Pernicious anemia.*

Anemia pseudoleukemica infantum, *May*, 1181
pernicious anemia and, differentiation, *Sept.*, 405
secondary, in latent gastric ulcer, *Jan.*, 766
splenic, splenectomy in, *Sept.*, 416
testing for, in protozoic enterocolitis, *May*, 1139
von Jaksch's, *May*, 1181
pernicious anemia and, differentiation, *Sept.*, 405
splenectomy in, *Sept.*, 416
Anemias, general classification, *May*, 1177
Anesthesia, surgical, in diabetes mellitus, *July*, 44
Aneurysm, abdominal, *Jan.*, 673
antisyphilitic treatment, *Jan.*, 678
history of case, *Jan.*, 673
physical examination, *Jan.*, 673
roentgen-ray examination in, *Jan.*, 676
treatment, *Jan.*, 678
of aorta, *March*, 1016
anginal pain in, *March*, 1024
characteristics of cough in, *March*, 1023
diagnosis, *March*, 1019
difficulty in swallowing in, *March*, 1025
etiology, *March*, 1023
history of case, *March*, 1016
hoarseness in, *March*, 1025
Huchard's sign in, *March*, 1019
physical examination, *March*, 1018
signs, *March*, 1026
pressure pains in, *March*, 1024
prognosis, *March*, 1028
symptomatology, *March*, 1023
syphilis in etiology, *March*, 1023
treatment, *March*, 1027
vertigo in, *March*, 1025
pathogenesis, *Jan.*, 677
skin pigmentation in, *Nov.*, 550
syphilis in etiology, *Jan.*, 677; *March*, 1023
Anginal pains in aneurysm of aorta, *March*, 1024
Anisocytosis in pernicious anemia, *Sept.*, 397
Antigonococcic vaccine in gonorrheal arthritis, *May*, 1195
Antisclerosin, *July*, 191
Aorta, aneurysm of, *March*, 1016. See also *Aneurysm of aorta.*
Aortic regurgitation, digitalis in, *July*, 5
stenosis, digitalis in, *July*, 5
Apex-beat in serofibrinous pericarditis, *March*, 931
Aphasia, auditory, *July*, 103
motor, *July*, 103
in recurrent endocarditis, *Nov.*, 596
varieties, *July*, 103
visual, *July*, 104
Aphemia, *July*, 103
Aplastic pernicious anemia, *Nov.*, 512
Appendicitis, chronic, chlorosis and, differentiation, *March*, 975
complicating duodenal ulcer, *Jan.*, 772
myelogenous leukemia and, differentiation, *Sept.*, 388
relation of, to chronic constipation, *March*, 1085
Apraxia in paraphasia, *July*, 99
Aregenerative anemia, *May*, 1180
Arhythmia, cardiac, *May*, 1199
juvenile, *May*, 1204
respiratory, *May*, 1204
sinus, *May*, 1204
varieties, *May*, 1203
Arneth and Leube's leukanemia, *May*, 1179
Arsenic in chlorosis, *March*, 984, 989
in pernicious anemia, *Nov.*, 519
Arteriosclerosis with diabetes mellitus, *July*, 13, 23
Arthritis, gonorrheal, *May*, 1189. See also *Gonorrheal arthritis.*
rheumatoid, anaphylaxis in, *March*, 869. See also *Anaphylaxis.*
Artificial feeding, *Sept.*, 249
albumin milk in, *Sept.*, 267
buttermilk mixtures in, *Sept.*, 268
caloric value of foods for, *Sept.*, 260
carbohydrates in, *Sept.*, 261
cereal pudding in, *Sept.*, 269
certified milk for, *Sept.*, 253
chymogen milk in, *Sept.*, 269
diluted milk in, *Sept.*, 258
Eiweiss milk in, *Sept.*, 267
energy value of foods in, *Sept.*, 259
examination of feces in, *Sept.*, 252
fats in, *Sept.*, 262
flour ball in, *Sept.*, 266
frequency, *Sept.*, 257
general principles, *Sept.*, 251
goats' milk in, *Sept.*, 256
Keller's malt soup in, *Sept.*, 268
larosan milk in, *Sept.*, 268
metabolism of salts in, *Sept.*, 262
pegnin milk in, *Sept.*, 269
percentage system, *Sept.*, 261
preparations for, *Sept.*, 266
protein cause of alimentary disturbances in, *Sept.*, 264
of cows' milk in, *Sept.*, 263
quantity at one time, *Sept.*, 256
relations of urine to, *Sept.*, 252
saccharin water in, *Sept.*, 266

Artificial feeding, tea in, *Sept.*, 266
whole milk in, *Sept.*, 258
Ascites, causes, *March*, 1044
differentiation of ailments associated with, *March*, 1052
fluids in, varieties, *May*, 1241
in carcinoma of hepatic flexure of colon, *May*, 1241
in retroperitoneal sarcoma, *March*, 1038
character of fluid, *March*, 1048, 1050
paracentesis for, *March*, 1047
prevention of recurrence, *March*, 1054
in syphilis of liver, *Sept.*, 247
Aspiration in serofibrinous pericarditis, *March*, 936
Astereognosis in pernicious anemia, *Nov.*, 520
Asthma from chronic constipation, *March*, 1084
Ataxic type of infantile paralysis, *Nov.*, 479
Atrophic type of starch injury in nutrition of infants, *Jan.*, 801
Atrophy in infants, *Jan.*, 805. See also *Decomposition.*
of optic nerve in tabes dorsalis, *May*, 1226
primary, tabes dorsalis and, differentiation, *May*, 1232
progressive muscular, *Sept.*, 289
Auditory aphasia, *July*, 103
Auricular extrasystole, *May*, 1205
fibrillation, *May*, 1208
digitalis in, *May*, 1210
pathology, *May*, 1209
flutter, *May*, 1210
pulse-wave, *May*, 1202
Auriculoventricular node, *May*, 1201
Auscultation in early pulmonary tuberculosis, *Jan.*, 747
Autogenous vaccines, action of, in oral infections, *March*, 992
Auto-intoxication and chronic constipation, *March*, 1080

Bacillus aërogenes capsulatus infection of intestines, *March*, 1007
elimination of organisms in, *March*, 1015
fecal fistulas after operation in, *March*, 1008
history of case, *March*, 1007
isolation of organism in, *March*, 1013
nature and course, *March*, 1013
Bacillus aërogenes capsulatus infection of intestines, operations in, *March*, 1007, 1008, 1011
treatment, dietetic, *March*, 1014
non-operative, *March*, 1011
Backache in tabes dorsalis, *May*, 1224
Bakers, prevalence of pulmonary diseases in, *May*, 1305
Banti's disease, splenectomy in, *Sept.*, 416
Barium in roentgen-ray examination of gastro-intestinal tract, *Jan.*, 819
sulphate medium in fluoroscopy, *July*, 209
test-meal for roentgen-ray examination, *Jan.*, 820
Basedow's disease, *July*, 85
apprehension in, *July*, 85
exophthalmos in, *July*, 86
history of case, *July*, 86
infected tonsils in etiology of, *July*, 87
skin pigmentation in, *Nov.*, 550
treatment, *July*, 88
Benzin in myelogenous leukemia, *Sept.*, 393
Benzol in myelogenous leukemia, *Sept.*, 393
Bier's hyperemia in gonorrheal arthritis, *May*, 1197
Bile acids and salts, Pettenkofer test for, *May*, 1095
Bile-pigments, research on chemistry of, *May*, 1093
tests for, *May*, 1094
in protozoic enterocolitis, *May*, 1138
Bilirubin, Gmelin test for, *May*, 1094
Bismuth in treatment of gastric ulcer, *Nov.*, 451
Bladder, papillomata of, *March*, 941. See also *Papillomata of bladder.*
tumors, cystography in diagnosis of malignancy, *March*, 945
Blaud's pill in chlorosis, *March*, 987
Blood, color-index of, in pernicious anemia, *Nov.*, 509
crises in pernicious anemia, *Sept.*, 402
examination in cardiospasm in dilatation of esophagus, *Nov.*, 617
in chlorosis, *March*, 976
in diabetes mellitus, *July*, 22
in latent gastric ulcer, *Jan.*, 766
in myelogenous leukemia, *Sept.*, 392
in pernicious anemia, *Sept.*, 409; *Nov.*, 512; *May*, 1176
in purpura hæmorrhagica, *Jan.*, 854
in rheumatoid arthritis, *March*, 870
in spasm at cardia in ulcus carcinomatosum, *Nov.*, 610

Blood examination in stenosis of pylorus with cholelithiasis, *Jan.*, 787
in feces in duodenal ulcer, *Jan.*, 772
transfusion in purpura hæmorrhagica, *Jan.*, 860
Blood-cells, red, Chauffard and Widal's test for fragility of, *May*, 1095
Blood-smears, technic of examination, *Sept.*, 395
Bone-marrow, carcinomatosis of, pernicious anemia and, differentiation, *Sept.*, 405; *May*, 1184
Bothriocephalus latus, hemolytic anemia from, *May*, 1179
Brain, changes in, in paralysis agitans, *March*, 950
Breast feeding, *July*, 114
cathartics in, *July*, 130
causes of failure in, *July*, 120
constitutional anomalies of infant in, *July*, 126
detoxication in, *July*, 128
diet of mother in, *July*, 117
establishment of, *July*, 116
excessive, *July*, 121
effects of, treatment, *July*, 122
exudative diathesis in, *July*, 126
for control of acute infections, *Jan.*, 797
immunity and, *July*, 118
in decomposition in infants, *Jan.*, 814
in lymphangitis, *July*, 120
in mastitis, *July*, 120
instructions to mother in, *July*, 117
insufficient, *July*, 122
causes, *July*, 123
diagnosis, *July*, 126
symptoms, *July*, 124
maternal conditions contra-indicating, *July*, 120
neuropathic diathesis in, *July*, 128
normal hunger of infant in, *July*, 123
nutritional disturbances in, *July*, 118
parenteral infections in infant in, *July*, 120
proper intervals in, *July*, 117
reparation period in, *July*, 129
septic infections during, *July*, 126
starvation in, *July*, 128
technic, *July*, 116
varieties, *July*, 116
milk, advantages of, for sick infants, 128
chemical examination, *July*, 118
colostrum and, comparison, *July*, 115
effects of emotions on, *July*, 121
of menstruation on, *July*, 121
Breast milk, establishing secretion of, *July*, 114
fat content of, *July*, 119
Breasts, maternal, varieties, *July*, 116
Bromid acne, *Sept.*, 319
Bronchiectasis, unilateral, *July*, 175
character of cavity formation in, *July*, 178
course, *July*, 175
differential diagnosis, *July*, 177
history of case, *July*, 175
onset, *July*, 175
physical examination, *July*, 176
putrid sputum in, *July*, 177
treatment, *July*, 179
Bronchitis, chronic, with emphysema, *Sept.*, 311
clubbed fingers in, *Sept.*, 312
cyanosis in, *Sept.*, 311
examination of abdomen in, *Sept.*, 315
of heart in, *Sept.*, 313
of lungs in, *Sept.*, 314
physical, *Sept.*, 311
roentgen-ray, *Sept.* 315
history of case, *Sept.*, 311
Bronchopneumonia, gangrene of lung from, *Nov.*, 562
low-grade, in recurrent endocarditis, *Nov.*, 602
Bruit du diable in chlorosis, *March*, 965
Bulbar type of infantile paralysis, *Nov.*, 479
Bundle of His, *May*, 1201
Buttermilk mixtures in artificial feeding, *Sept.*, 268

Cachectic purpura in infants, *Jan.*, 807
Caffein with digitalis, *July*, 10
Caloric value of foods for artificial feeding, *Sept.*, 260
Canadian hemp with digitalis, *July*, 10
Cancer. See *Carcinoma.*
Cancerous conditions, skin pigmentation in, *Nov.*, 550
Caput medusæ in cirrhosis of liver, *May*, 1251
Carbohydrates in artificial feeding, *Sept.*, 261
Carcinoma of esophagus, *May*, 1143
complications, *May*, 1152
diagnosis, *May*, 1153
differential, *May*, 1153
difficulty in swallowing in, *May*, 1151
esophagoscope in, *May*, 1154
etiology, *May*, 1148
histories of cases, *May*, 1144, 1146
location, *May*, 1150
metastases from, *May*, 1151

Carcinoma of esophagus, pain in, *May*, 1152
pathology, *May*, 1149
physical examination, *May*, 1144, 1147
prognosis, *May*, 1155
regurgitation of food in, *May*, 1151
roentgen-ray examination, *May*, 1153
sites of predilection, *May*, 1150
size, *May*, 1150
symptomatology, *May*, 1151
treatment, *May*, 1155
dietetic, *May*, 1156
mechanical, *May*, 1156
palliative, *May*, 1155
radium, *May*, 1158
surgical, *May*, 1158
varieties, *May*, 1149
vomiting in, *May*, 1151
of gall-bladder, pancreatic carcinoma and, differentiation, *Sept.*, 307
of head of pancreas, *Sept.*, 303
blood examination, *Sept.*, 304
differential diagnosis, *Sept.*, 307
history of case, *Sept.*, 303
jaundice in, *Sept.*, 303
laboratory findings, *Sept.*, 304
physical examination, *Sept.*, 304
roentgen-ray examination in, *Sept.*, 305
treatment, *Sept.*, 310
of hepatic flexure of colon, *May*, 1237
absence of pain in, *May*, 1243, 1249
ascites in, *May*, 1241
diagnosis, *May*, 1248
edema in, *May*, 1237
history of case, *May*, 1237
paracentesis in, *May*, 1242
physical examination, *May*, 1240
roentgen-ray examination in, *May*, 1243
of pancreas, gall-bladder carcinoma and, differentiation, *Sept.*, 307
of rectum, *Jan.*, 731
colostomy in, *Jan.*, 743
constipation in, *Jan.*, 738
diagnosis, *Jan.*, 741
differential diagnosis, *Jan.*, 741
digital examination, *Jan.*, 740
etiology, *Jan.*, 737
general considerations, *Jan.*, 736
hemorrhage in, *Jan.*, 739
hemorrhoids in etiology, *Jan.*, 738
history of case, *Jan.*, 730, 734
instrumental examination, *Jan.*, 740
location, *Jan.*, 737
Carcinoma of rectum, non-malignant strictures and, differentiation, *Jan.*, 741
objective symptoms, *Jan.*, 740
occurrence, *Jan.*, 737
operation, *Jan.*, 733, 735
pain in, *Jan.*, 739
physical examination, *Jan.*, 732, 734
subjective symptoms, *Jan.*, 738
symptoms, *Jan.*, 731, 734, 738
syphilis and, differentiation, *Jan.*, 741
treatment, palliative, *Jan.*, 742
radium in, *Jan.*, 743
roentgen rays in, *Jan.*, 743
surgical, *Jan.*, 742
varieties, *Jan.*, 736
pernicious anemia and, differentiation, *May*, 1184
Carcinomatosis of bone-marrow, pernicious anemia and, differentiation, *Sept.*, 405; *May*, 1184
Cardia, spasm at, *Nov.*, 607
cardiospasm and, differentiation, *Nov.*, 607
in benign ulcer, *Nov.*, 611
fluoroscopic examination, *Nov.*, 612
history of case, *Nov.*, 611
physical examination, *Nov.*, 611
test-meal in, *Nov.*, 611
treatment, *Nov.*, 612
in ulcus carcinomatosum, *Nov.*, 608
blood examination in, *Nov.*, 610
fluoroscopic examination in, *Nov.*, 610
history of case, *Nov.*, 608
physical examination, *Nov.*, 609
treatment, *Nov.*, 610
in vagotonia, *Nov.*, 613
history of case, *Nov.*, 613
physical examination, *Nov.*, 613
treatment, *Nov.*, 614
Cardiac arhythmia, *May*, 1199. See also *Arhythmia, cardiac.*
contraction, premature, *May*, 1203
Cardiospasm, *Nov.*, 607
absence of pain in, *Nov.*, 620
after-treatment, *Nov.*, 630
age incidence, *Nov.*, 619
constipation in, *Nov.*, 621
diagnosis, *Nov.*, 619
dysphagia in, *Nov.*, 620
from dysphagia with dilatation of esophagus, *Nov.*, 615
blood examination, *Nov.*, 617
fluoroscopic examination, *Nov.*, 618
history of case, *Nov.*, 615

Cardiospasm from dysphagia with dilatation of esophagus, physical examination, *Nov.*, 616
reflexes in, *Nov.*, 617
permanence of cure in, *Nov.*, 630
preparation of patient for esophageal instrumentation in, *Nov.*, 627
rectal feeding in, *Nov.*, 630
regurgitation in, *Nov.*, 620
roentgen-ray examination in, *Nov.*, 621
sex and, *Nov.*, 619
spasm at cardia and, differentiation, *Nov.*, 607
strength loss in, *Nov.*, 621
treatment, instruments for, *Nov.*, 627
Plummer's apparatus for, *Nov.*, 627–629
vomiting in, *Nov.*, 620
weight loss in, *Nov.*, 620
Carotid pulse wave, *May*, 1202
Carriers of protozoa, *May*, 1110
Cascara in chlorosis, *March*, 989
Catarrh, postgonorrheal, in specific urethritis, *March*, 1067
Catarrhal jaundice, illustrative case, *May*, 1102
Catatonia of involution, *Jan.*, 714
Cathartics in infants, *July*, 130
in chronic constipation, *March*, 1088
Cercomonads in protozoic enterocolitis, *May*, 1114, 1118
Cercomonas intestinalis hominis in protozoic enterocolitis, *May*, 1133
Cereal pudding in artificial feeding, *Sept.*, 269
Cerebral centers, development of association pathways of, *July*, 105
localization of, *July*, 104
embolism in recurrent endocarditis, *Nov.*, 597
Cerebrospinal fluid in general paresis, *July*, 67
meningitis, *July*, 193
diagnosis, *July*, 196
Flexner's serum in, *July*, 197
history of case, *July*, 193
Kernig sign in, *July*, 195
physical examination, *July*, 194
treatment, *July*, 197
Certified milk, *Sept.*, 253
chemistry of, *Sept.*, 255
requirements for production of, *Sept.*, 254
Cesarean section in diabetes mellitus, *July*, 49
Chauffard and Widal's test for fragility of red blood-cells, *May*, 1095
Chemical achylia, *Jan.*, 700
Chlorosis, *March*, 963; *May*, 1180
arsenic in, *March*, 984, 989
Blaud's pill in, *March*, 987
blood examination in, *March*, 976
bruit du diable in, *March*, 965
cascara in, *March*, 989
chronic appendicitis and, differentiation, *March*, 975
diagnosis, *March*, 978
duodenal ulcer and, differentiation, *March*, 972
Fowler's solution in, *March*, 989
gastric analysis in, *March*, 972
ulcer and, differentiation, *March*, 972
Grawitz's theory, *March*, 982
history of case, *March*, 963
hyperthyroidism and, differentiation, *March*, 975
iron in, *March*, 984
by subcutaneous injection, *March*, 987
Morawitz's theory, *March*, 982
Müller's theory, *March*, 982
Naegeli's grouping, *March*, 979
physical examination, *March*, 965
pulmonary tuberculosis and, differentiation, *March*, 969
roentgen-ray examination of gastrointestinal tract in, *March*, 974
status hypoplasticus in, *March*, 982
symptomatology, *March*, 980
symptoms, *March*, 964
theories of etiology, *March*, 981
treatment, *March*, 984
tuberculin tests in, *March*, 970
types, *March*, 979
urinalysis in, *March*, 977
Virchow's theory, *March*, 981
von Noorden's theory, *March*, 983
Cholecystitis complicating duodenal ulcer, *Jan.*, 770
Cholelithiasis and pyloric stenosis, *Jan.*, 785. See also *Pylorus, stenosis of, cholelithiasis and.*
Cholemia, *May*, 1092
Chymogen milk in artificial feeding, *Sept.*, 269
Cirrhosis of liver, caput medusæ in, *May*, 1251
retroperitoneal sarcoma and, differentiation, *March*, 1045
syphilis of liver and, differentiation, *Sept.*, 242
syphilitic, *May*, 1278. See also *Syphilitic cirrhosis of liver.*
Claudication, intermittent, *July*, 187
blood-examination in, *July*, 18
history, *July*, 187
physical examination, *July*, 187

Claudication, intermittent, prognosis, *July*, 191
treatment, *July*, 191
Closed pneumothorax, *May*, 1297
Clubbed fingers in chronic bronchitis, with emphysema, *Sept.*, 312
Cole's serial method of roentgenography, *July*, 218
Coley's serum in retroperitoneal sarcoma, *March*, 1055
Colicystitis of Escherich, *March*, 898
Colitis, catarrhal, chronic constipation in, *March*, 1085
mucous, associated with chronic constipation, *March*, 1086
dietetic treatment, *Sept.*, 357
irrigation treatment, *Sept.*, 356
ulcerative, topical applications in, *Sept.*, 356
Colon bacillus, pyelitis from, treatment, *Nov.*, 567
diverticulitis of, from chronic constipation, *March*, 1085
hepatic flexure of, carcinoma of, *May*, 1237. See also *Carcinoma of hepatic flexure of colon.*
in splanchnoptosis, *Jan.*, 642
mass movement in, *Jan.*, 826
Colostomy in carcinoma of rectum, *Jan.*, 743
Colostrum, *July*, 114
and breast milk, comparison, *July*, 115
functions of, *July*, 115
Comedones, removal of, in acne, *Sept.*, 323
Complement fixation test in gonorrheal arthritis, *May*, 1193
Conduction system of heart, *May*, 1199
Constipation, avoidance of, in treatment of chronic diarrhea, *Sept.*, 356
chronic, *March*, 1071
anal fissure from, *March*, 1080
associated pathologic conditions, *March*, 1084
asthma from, *March*, 1084
atonic type, *March*, 1075
auto-intoxication and, *March*, 1080
classification, *March*, 1073
diverticulitis of colon from, *March*, 1085
dyschezia in, *March*, 1078
treatment, *March*, 1089
from delay in passage of feces through intestines, *March*, 1074
from depression of nervous system, *March*, 1075
from habitual neglect, *March*, 1078
from inefficient muscles of defecation, *March*, 1079
Constipation, chronic, from insufficient food, *March*, 1075
from lack of exercise, *March*, 1075
from Lane's kinks, *March*, 1078
hemorrhoids from, *March*, 1080
in catarrhal colitis, *March*, 1085
in visceroptosis, *March*, 1084
kidney infections from, *March*, 1084
local effects of, *March*, 1080
mucous colitis associated with, *March*, 1086
pathologic conditions associated with, *March*, 1084
physiologic considerations, *March*, 1071
prophylaxis, *March*, 1087
relation of gastric secretion to, *March*, 1081
to appendicitis, *March*, 1085
to Hirschsprung's disease, *March*, 1086
to intestinal flatulence, *March*, 1083
to megacolon, *March*, 1086
spastic, *March*, 1076
treatment, *March*, 1090
symptomatology, *March*, 1079
treatment, abdominal belt in, *March*, 1090
cathartics in, *March*, 1088
dietetic, *March*, 1087
enemas in, *March*, 1089
massage in, *March*, 1090
mechanical, *March*, 1089
medicinal, *March*, 1088
of neurasthenia in, *March*, 1090
purgatives in, *March*, 1088
surgical, *March*, 1090
in carcinoma of rectum, *Jan.*, 738
in cardiospasm, *Nov.*, 621
in duodenal ulcer, *Jan.*, 780
in protozoic enterocolitis, *May*, 1125
in splanchnoptosis, *Jan.*, 642
Cows' milk, certified. See *Certified milk.*
diluted, in artificial feeding, *Sept.*, 258
fermented, in treatment of chronic diarrhea, *Sept.*, 356
protein of, in artificial feeding, *Sept.*, 263
whole, in artificial feeding, *Sept.*, 258
Creosote bath in gangrene of lung, *Nov.*, 563
Crypts of Morgagni, involvement of, in specific urethritis, *March*, 1062
Cyanosis in chronic bronchitis with emphysema, *Sept.*, 311
Cyclic vomiting, treatment, *July*, 63
Cystography for determining malignancy of bladder tumors, *March*, 945

Decomposition in infants, *Jan.*, 805
acidosis in, treatment, *Jan.*, 817
albumin milk in, *Jan.*, 815
breast feeding in, *Jan.*, 814
clinical course, *Jan.*, 812
dangers of therapeutic starvation, *Jan.*, 812
demineralization in, *Jan.*, 813
diarrhea in etiology, *Jan.*, 808
treatment, *Jan.*, 817
etiology, *Jan.*, 805
feces in, *Jan.*, 811
history of case, *Jan.*, 805
improper diet in etiology, *Jan.*, 809
physical examination, *Jan.*, 806
reparation stage in, *Jan.*, 815
subnormal temperature in, *Jan.*, 810
symptomatology, *Jan.*, 809
treatment, *Jan.*, 814
underfeeding in, etiology, *Jan.*, 809
Defecation, mechanism of, *March*, 1072
Demineralization in decomposition in infants, *Jan.*, 813
Deodorants, dermatitis from, *Nov.*, 539
Dermatitis from deodorants, *Nov.*, 539
hair-dye, cases of, *Nov.*, 538, 539
mange-cure, *Nov.*, 540
medicamentosa, *Sept.*, 319
occupational, acquired susceptibility in, *Nov.*, 543
treatment, *Nov.*, 544
orthoform, cases of, *Nov.*, 535, 537
paraphenylendiamin, cases of, *Nov.*, 538, 539
pigment, *Nov.*, 541
primrose, cases of, *Nov.*, 533, 534
Detoxication in infants, *July*, 128
Diabetes innocens of young, *Sept.*, 340
mellitus, *July*, 13
acidosis in, dietetic treatment, *July*, 51
additional notes on cases of, *July*, 26
alkali therapy in, *July*, 51
Allen treatment, inanition in, *March*, 999
keeping urine sugar free in, *March*, 1005
limitations, *March*, 1004
origin and development of, *March*, 1001
period of observation before, *March*, 1004
raising carbohydrate tolerance in, *March*, 1006
rendering urine sugar free in, *March*, 1004
results from, *March*, 1003
starvation period, *March*, 1002, 1003
Diabetes mellitus, Allen treatment, strenuous exercise in, *March*, 1003
technic, *March*, 1002
arteriosclerosis in, *July*, 13, 23
blood examination in, *July*, 22
chronic peripheral neuritis in, *July*, 13, 23
dangers from surgical operation in, *July*, 41
delayed, *March*, 1000
diagnosis, *July*, 19, 20, 23, 24, 26
dietary instructions to patient in, *Sept.*, 331
from extirpation of pancreas, *March*, 1000
histories of cases, *July*, 13, 19, 21, 23, 25
importance of early treatment, *Sept.*, 330
in young, *Sept.*, 327
after-treatment, *Sept.*, 332
Allen treatment, *Sept.*, 328, 334
diagnosis, *Sept.*, 341
histories of cases, *Sept.*, 327, 332, 333, 336
subsequent notes on, *Sept.*, 338
prognosis, *Sept.*, 337
myocarditis with, *July*, 23
origin of dietetic treatment, *March*, 1001
physical examination in, *July*, 14, 20, 25
pregnancy in, *July*, 47
Cesarean section in, *July*, 49
dietetic treatment, *July*, 49
history of case, *July*, 47
preparing patient for operation in, *July*, 42
report of surgical operations in, *July*, 45
splanchnoptosis with, *July*, 23
surgical anesthesia in, *July*, 44
operation in, *July*, 37, 41
history of case, *July*, 37
physical examination in, *July*, 37, 41
temperature in, *July*, 22
tendency to gangrene in, *July*, 19
urinalysis in, *July*, 15, 20, 21, 24, 26
with acidosis, *July*, 29
decreasing fats in diet, *July*, 35
dietetic treatment, *July*, 35
further report on, *July*, 36
general considerations, *July*, 33
history of case, *July*, 29
laboratory findings, *July*, 31
medicinal treatment, *July*, 35
physical examination, *July*, 30

Diabetes mellitus with acidosis, temperature in, *July*, 30
with chronic interstitial nephritis, *July*, 13
with gangrene of toes, *July*, 19, 23
with pulmonary tuberculosis, *July*, 21
renal, pregnancy in, *July*, 50
Diarrhea, chronic, *Sept.*, 343
appearance of intestinal mucosa in, *Sept.*, 347
differentiation of types of, *Sept.*, 345
examination of stomach contents in, *Sept.*, 344, 350
of stools in, *Sept.*, 345, 349
gastrogenic, diagnosis, *Sept.*, 351
prognosis, *Sept.*, 351
histories of cases, *Sept.*, 343, 348, 352
physical examination, *Sept.*, 343, 348, 353
proctoscopic examination, *Sept.*, 344
prognosis, *Sept.*, 348
roentgen-ray examination in, *Sept.*, 351
theories of causes, *Sept.*, 346
treatment, *Sept.*, 354
avoidance of constipation in, *Sept.*, 356
dietetic, *Sept.*, 354
drugs in, *Sept.*, 357
local, *Sept.*, 356
surgical, *Sept.*, 357
with fermented milk, *Sept.*, 356
gastrogenic, in achylia gastrica, *Jan.*, 702
in decomposition in infants, treatment, *Jan.*, 817
in etiology of decomposition in infants, *Jan.*, 808
in protozoic enterocolitis, *May*, 1124
Diarsenol in pernicious anemia, *Nov.*, 531
Diathesis, exudative, in infants, *Jan.*, 797
Digalen, *July*, 8
Digipuratum, *July*, 8
Digitalin, *July*, 8
Digitalis, *July*, 1
adjuvants, *July*, 9
administration of, *July*, 7
advantages of tincture, *July*, 8
caffein with, *July*, 10
Canadian hemp with, *July*, 10
contra-indications to, *July*, 5
cumulative action of, *July*, 7
diuretin with, *July*, 10
dyspnea in, *July*, 11
Digitalis, gastro-intestinal symptoms from, *July*, 6
general indications for, *July*, 3
in anemia, *July*, 11
in aortic regurgitation, *July*, 5
stenosis, *July*, 5
in auricular fibrillation, *May*, 1210
in dilation of heart, *July*, 3
in dropsy, *July*, 9
in dyspnea, *July*, 11
in gastric symptoms, *July*, 12
in heart-block, *May*, 1213
in insomnia, *July*, 11
in irregularity of heart, *July*, 3
in mitral stenosis, *July*, 5
in pain, *July*, 12
in palpitation, *July*, 12
in paroxysmal tachycardia, *May*, 1215
in pulsus alternans, *May*, 1212
in respiratory symptoms, *July*, 11
in valvular lesions of heart, *July*, 5
in weak heart, *July*, 3
morphin with, *July*, 11
preparations of, *July*, 7
rapid heart in, *July*, 3
substitutes for, *July*, 8
sweats with, *July*, 1
symptomatic therapy, *July*, 9
toxic manifestations of, *July*, 6
use of, *July*, 1
with free purgation, *July*, 6
with Karell diet, *July*, 6
with rest in bed, *July*, 6
with sweats, *July*, 10
with venesection, *July*, 6
Digitoxin, *July*, 8
Dilatation of heart, digitalis in, *July*, 3
Dissociation jaundice in pernicious anemia, illustrative case, *May*, 1106
Diuretin with digitalis, *July*, 10
Diverticulitis of colon from chronic constipation, *March*, 1085
Dropped pulse beat, *May*, 1207
Dropsy, deep incision in, *July*, 10
digitalis in, *July*, 9
Drugs, skin pigmentation from, *Nov.*, 550
Duodenal bulb in fluoroscopy of stomach, *July*, 213
cap, defective, in duodenal ulcer, *Nov.*, 436, 436
normal, roentgen-ray appearance, *Nov.*, 433, 434
ulcer, *Jan.*, 477; *Nov.*, 419
attempt at perforation in, *Jan.*, 782
blood in feces in, *Jan.*, 772
chlorosis and, differentiation, *March*, 972
complicated, *Jan.*, 769
diagnosis, *Jan.*, 773

Duodenal ulcer, complicated, examination, *Jan.*, 770
history of case, *Jan.*, 769
prognosis after operation, *Jan.*, 775
pulmonary infarct in, *Jan.*, 773
treatment, *Jan.*, 774
with appendicitis, *Jan.*, 772
with cholecystitis, *Jan.*, 770
conclusions on, *Nov.*, 437
constipation in, *Jan.*, 780
defective duodenal cap in, *Nov.*, 435, 436
examination in, *Jan.*, 779
gastric ulcer and, differentiation, *Jan.*, 764
gastrojejunostomy in, *Jan.*, 783
headache in, *Jan.*, 781
history of case, *Jan.*, 777
myelogenous leukemia and, differentiation, *Sept.*, 388
phrenic nerve neuralgia in, *Jan.*, 782
roentgen-ray examination, value of, *Nov.*, 438
sciatic neuritis in, *Jan.*, 780
treatment, surgical, *Jan.*, 783
Duodenum, ulcer of. See *Duodenal ulcer.*
Dysbasia angiosclerotica, *July*, 189
Dyschezia, *March*, 1073
in chronic constipation, *March*, 1078, 1089
Dysentery, amebic, *Jan.*, 679. See also *Amebic dysentery.*
Dyspepsia in protozoic enterocolitis, *May*, 1126
nervous, in splanchnoptosis, *Jan.*, 641, 642
Dysphagia in cardiospasm, *Nov.*, 620
with dilatation of esophagus, cardiospasm from, *Nov.*, 615
Dyspnea, digitalis in, *July*, 11
Dysthyroidism as cause of manic-depressive insanity, *July*, 78

Eczema from external irritation, *Nov.*, 533
acquired susceptibility in, *Nov.*, 543
treatment, *Nov.*, 544
Edema, causes of, *May*, 1238
in carcinoma of hepatic flexure of colon, *May*, 1237
Ehrlich aldehyd reaction for urobilinogen, *May*, 1094
Einhorn's gastrodiaphane in splanchnoptosis, *Jan.*, 644
Eiweiss milk in artificial feeding, *Sept.*, 267
Electricity in infantile paralysis, *Nov.*, 485, 507
Embolism, causes of, *July*, 99
cerebral, in recurrent endocarditis, *Nov.*, 597
origin of, *July*, 101
Emetin hydrochlorid in amebic dysentery, *Jan.*, 680
Emotions, effects of, on breast milk, *July*, 120
Emphysema, pneumothorax due to, *May*, 1291. See also *Pneumothorax due to emphysema.*
with chronic bronchitis, *Sept.*, 311
Encephalitis type of infantile paralysis, *Nov.*, 479
Endocardial murmur in serofibrinous pericarditis, *March*, 931
Endocarditis in hemorrhagic pericarditis, *March*, 924
miliary tuberculosis and, differentiation, *Sept.*, 228
recurrent, *Nov.*, 593
broken compensation in, *Nov.*, 601
cerebral embolism in, *Nov.*, 597
diagnosis, *Nov.*, 602
further report on, *Nov.*, 598
hemoptysis in, *Nov.*, 601
history of case, *Nov.*, 593
low-grade bronchopneumonia in, *Nov.*, 602
motor aphasia in, *Nov.*, 596
physical examination, *Nov.*, 593
reflexes in, *Nov.*, 595
right-sided hemiplegia in, *Nov.*, 596
splenic infarct in, *Nov.*, 600
thrombo-ulcerative, *July*, 181
autopsy findings, *July*, 185
course, *July*, 183
hemolytic jaundice in, *July*, 183
hemorrhage in, *July*, 183
history of case, *July*, 181
regional examination, *July*, 181
termination, *July*, 183
Enema, opaque, fluoroscopic examination after injection of, *Jan.*, 834
technic of injection, *Jan.*, 832
Entameba histolytica and Entameba coli, differentiation, *May*, 1135
in protozoic enterocolitis, *May*, 1114
Entamebæ in protozoic enterocolitis, description, *May*, 1133
Enterocolitis, protozoic, *May*, 1109. See also *Protozoic enterocolitis.*
Epistaxis in myelogenous leukemia, *Sept.*, 391
Erb's dysbasia angiosclerotica, *July*, 189
Escherich's colicystitis, *March*, 898
Esophagoscope in carcinoma of esophagus, *May*, 1154

Esophagus, carcinoma of, *May*, 1143. See *Carcinoma of esophagus.*
dilatation of, with dysphagia, cardiospasm from, *Nov.*, 615
fluoroscopy of, *July*, 210
Exercise in treatment of splanchnoptosis, *Jan.*, 646
lack of, chronic constipation from, *March*, 102
Exner's writing center, *July*, 104
Exophthalmic goiter, *July*, 85. See also *Basedow's disease.*
Exophthalmos in Basedow's disease, *July*, 86
Extrasystole, *May*, 1204
auricular, *May*, 1205
ventricular, *May*, 1205
Exudates in ascites, *May*, 1241
Exudative diathesis in infants, *July*, 126; *Jan.*, 797
Eyelids, ptosis of, in tabes dorsalis, *May*, 1220

Fat content of breast milk, *July*, 119
decreasing of, in diet in diabetes mellitus with acidosis, *July*, 35
feeding of infants, relation of, to resistance and nutritional disturbance, *Jan.*, 799
in artificial feeding, *Sept.*, 262
Fecal fistulas after operation in Bacillus aërogenes capsulatus infection of intestines, *March*, 1008
Feces, blood in, in duodenal ulcer, *Jan.*, 772
color of, in latent gastric ulcer, *Jan.*, 762
examination of, in artificial feeding, *Sept.*, 252
in chronic diarrhea, *Sept.*, 345
in protozoic enterocolitis, *May*, 1129, 1136, 1137
in decomposition in infants, *Jan.*, 811
Feeding, artificial, *Sept.*, 249. See also *Artificial feeding.*
breast, *July*, 114. See also *Breast feeding.*
normal infant, *July*, 113
Fermented milk treatment of chronic diarrhea, *Sept.*, 356
Fever in syphilis of liver, *Sept.*, 247
Fibrillation, auricular, *May*, 1208
digitalis in, *May*, 1210
pathology, *May*, 1210
Fibroid induration of lungs, *May*, 1261
Fish-poisoning, anaphylaxis in, *March*, 865. See also *Anaphylaxis.*
Flack and Keith's node, *May*, 1200
Flexner and Amoss' immune serum in infantile paralysis, *Nov.*, 484
Flexner's serum in cerebrospinal meningitis, *July*, 197
in infantile paralysis, *Nov.*, 507
Flour ball in artificial feeding, *Sept.*, 266
Fluorescent screen, description, *July*, 200
Fluoroscopic appearance of stomach, *July*, 211
examination after injection of opaque enema, *Jan.*, 833
in cardiospasm in dilatation of esophagus, *Nov.*, 618
in incipient pulmonary tuberculosis, *Jan.*, 753
in spasm at cardia, in benign ulcer, *Nov.*, 612
in ulcus carcinomatosum, *Nov.*, 610
in vagotonia, *Nov.*, 614
Fluoroscopy, dangers to operator in, *July*, 210
in upright position, *July*, 207
leaded gloves for operator in, *July*, 206
of chest, diagnostic value, *July*, 208
of esophagus, *July*, 210
of stomach, *July*, 199
air bubble in, *July*, 209
barium sulphate medium for, *July*, 209
duodenal bulb in, *July*, 213
Haudek's double-meal method, *July*, 220
horizontal, *July*, 214
peristaltic waves in, *July*, 212
value of, *July*, 205
palpation underneath screen in, *July*, 204
preparation of operator's eyes for, *July*, 199
protecting patient during, *July*, 202
roentgenography and, comparative value, *July*, 204
Flutter, auricular, *May*, 1211
Food, insufficient, constipation from, *March*, 1075
regurgitation, vomiting and, differentiation, *July*, 56
Fowler's solution in chlorosis, *March*, 989
Fränkel's exercises in tabes dorsalis, *May*, 1235
Fridericia method of estimating acidosis, *Jan.*, 683
Fulguration of papillomata of bladder, *March*, 942, 943
local reaction from, *March*, 948
number of treatments required, *March*, 947
pain in, *March*, 946
results from, *March*, 947
selection of cases for, *March*, 945

Fulguration of papillomata of bladder, technic, *March*, 944
where to begin, *March*, 946

Gait in tabes dorsalis, *May*, 1217, 1225, 1226
Gangrene of lung, *Nov.*, 557
causes, *Nov.*, 562
creosote bath in, *Nov.*, 563
etiology, *Nov.*, 561
history of case, *Nov.*, 557
lobar pneumonia and, differentiation, *Nov.*, 560
physical examination, *Nov.*, 557, 558
prognosis, *Nov.*, 563
pulmonary tuberculosis and, differentiation, *Nov.*, 559
roentgen-ray examination in, *Nov.*, 565
surgical drainage in, *Nov.*, 564
treatment, *Nov.*, 563
of toes in diabetes mellitus, *July*, 19, 25
tendency to, in diabetes mellitus, *July*, 13
Gastric analysis in chlorosis, *March*, 972
in protozoic enterocolitis, *May*, 1138
emptying time in achylia gastrica, *Jan.*, 701
motility in achylia gastrica, *Jan.*, 700
secretion, relation of, to constipation, *March*, 1081
symptoms, digitalis in, *July*, 12
ulcer, *Nov.*, 419
apparatus for obtaining chymograph tracings in, *Nov.*, 443
benign, spasm at cardia in, *Nov.*, 611
bismuth treatment, *Nov.*, 451
chlorosis and, differentiation, *March*, 972
chronic, absolute rest in bed in, *Nov.*, 453
beginning feeding in, *Nov.*, 455
diet in, *Nov.*, 455
differential diagnosis, *Nov.*, 452
drug treatment, *Nov.*, 457
enemas in, *Nov.*, 454, 457
management after discharge from hospital, *Nov.*, 458
medical treatment, *Nov.*, 452
versus surgical treatment, *Nov.*, 460
observation, *Nov.*, 452
starvation treatment, *Nov.*, 453
surgical treatment versus medical treatment, *Nov.*, 460
use of stomach-tube in, *Nov.*, 461
chymograph tracings from, *Nov.*, 445–449
Gastric ulcer, conclusions on, *Nov.*, 437
diagnosis, *Nov.*, 422
epigastric distress in, *Nov.*, 441
etiology, *Nov.*, 438
experimental, in dogs, findings in, *Nov.*, 440
explanation of pain mechanism in, *Nov.*, 442
free hydrochloric acid as cause, *Nov.*, 441
histories of cases, *Nov.*, 419, 432
hunger pains in, *Nov.*, 441, 442
increased tension on walls of stomach as cause, *Nov.*, 442
laboratory findings, *Nov.*, 420
latent, *Jan.*, 761
blood examination in, *Jan.*, 766
color of feces in, *Jan.*, 762
duodenal ulcer and, differentiation, *Jan.*, 764
examination in, *Jan.*, 762
hemorrhage in, *Jan.*, 761
history of case, *Jan.*, 761
secondary anemia in, *Jan.*, 766
treatment, *Jan.*, 767
dietetic, *Jan.*, 768
urinalysis in, *Jan.*, 766
mechanically delayed emptying time in, *Nov.*, 438
medical cure of, *Nov.*, 427
positive evidences, *Nov.*, 428
treatment, *Nov.*, 424, 432, 451
results of, *Nov.*, 424, 434
motor insufficiency in etiology, *Nov.*, 439
myelogenous leukemia and, differentiation, *Sept.*, 387
neutralization of free HCl in, *Nov.*, 461
physical examination, *Nov.*, 420
possibilities of medical cure in, *Nov.*, 422
roentgen-ray examination in, *Nov.*, 422
after medical treatment, *Nov.*, 425
value of, *Nov.*, 427
surgical treatment, *Nov.*, 431
symptomatology, *Nov.*, 441
vomiting in, *Nov.*, 420
Gastritis, acute, vomiting from, *July*, 50
Gastrodiaphane, Einhorn's, in splanchnoptosis, *Jan.*, 644
Gastrogenic diarrhea in achylia gastrica, *Jan.*, 702
Gastro-intestinal tract, roentgen-ray examination, cleansing enemas before, *Jan.*, 821
in chlorosis, *March*, 974
laxatives before, *Jan.*, 845

Gastro-intestinal tract, roentgen-ray examination, opaque enema for, *Jan.*, 822
time for passage of opaque meal through, *Jan.*, 828
Gastroptosis in achylia gastrica, *Jan.*, 702
Gaucher's type of splenomegaly, splenectomy in, *Sept.*, 416
Gibson and Hirschfelder's pulse wave, *May*, 1203
Glénard's disease, *Jan.*, 631. See also *Splanchnoptosis.*
Glycosuria, alimentary, *Sept.*, 340
renal, *Sept.*, 339
history of case, *Sept.*, 339
treatment, *Sept.*, 340
Gmelin test for bilirubin, *May*, 1094
Goats' milk for artificial feeding, *Sept.*, 256
Goiter, exophthalmic, *July*, 85. See also *Basedow's disease.*
Goldscheider's orthopercussion in pulmonary tuberculosis, *March*, 966
Gonococcus, detection of, in specific urethritis, *March*, 1059
method of staining, *March*, 1060
Gonorrhea, painful heel of, *May*, 1192
Gonorrheal arthritis, *May*, 1189
acute gonococcus, *May*, 1191
arthralgic, *May*, 1191
bursal, *May*, 1192
complement fixation test in, *May*, 1193
complications, *May*, 1192
diagnosis, *May*, 1193
history of case, *May*, 1190
Osler's classification, *May*, 1191
polyarthritic, *May*, 1191
prophylaxis, *May*, 1194
prostatic abscess in, *May*, 1191
synovial, *May*, 1192
treatment, *May*, 1194
antigonococcic vaccine, *May*, 1195
general, *May*, 1195
hyperemic, *May*, 1197
of urethral infections in, *May*, 1194
specific, *May*, 1195
hydroarthrosis, chronic, *May*, 1192
urethritis, *March*, 1057. See also *Urethritis, specific.*
Gout, atypical, *Nov.*, 603
acute articular rheumatism simulating, *Nov.*, 604
chronic nephritis in, *Nov.*, 605
examination of tophus in, *Nov.*, 604
history of case, *Nov.*, 603
physical examination, *Nov.*, 603
Grawitz's theory of chlorosis, *March*, 982
Gumma formation in syphilitic infection of lungs, *May*, 1261
in syphilis of liver, *Sept.*, 243
Guyon's capillary syringe, *March*, 1063

Hair-dye dermatitis, cases of, *Nov.*, 538, 539
Haudek's double-meal method of fluoroscopy of stomach, *July*, 220
Headache in duodenal ulcer, *Jan.*, 781
Heart compensation, broken, *July*, 2
conduction system of, *May*, 1199
dilatation of, digitalis in, *July*, 3
diseases, classification of, *July*, 1
digitalis in, *July*, 1
findings in hemorrhagic pericarditis, *March*, 919
in purulent pericarditis, *March*, 910
hypertrophy of, *July*, 2
insufficiency of, digitalis in, *July*, 5
irregularity of, digitalis in, *July*, 3
lesions, valvular, digitalis in, *July*, 5
rapid, digitalis in, *July*, 3
reserve power of, *July*, 2
weak, digitalis in, *July*, 3
Heart-block, *May*, 1212
treatment, *May*, 1213
Hedinger-Schlayer renal test, Mosenthal's modification, *March*, 1029
Heel, painful, of gonorrhea, *May*, 1192
Hematemesis, *May*, 1265
differential diagnosis, *May*, 1267
hemoptysis simulating, *May*, 1284. See also *Hemoptysis simulating hematemesis.*
in pernicious anemia, *May*, 1286
history of case, 1286
physical examination, *May*, 1287
in syphilitic cirrhosis of liver, *May*, 1265, 1280
anatomic diagnosis, *May*, 1277
histologic report, *May*, 1276
history of case, *May*, 1265
physical examination, *May*, 1266
postmortem findings, *May*, 1271
sources of bleeding in, *May*, 1289
Hematogenous jaundice, illustrative case, *May*, 1104
Hematuria in papillomata of bladder, *March*, 942, 943
Hemiplegia, right-sided, in recurrent endocarditis, *Nov.*, 596
Hemoglobin, overproduction of, in pernicious anemia, *Nov.*, 510
Hemolytic anemia from Bothriocephalus latus, *May*, 1179
relation of syphilis to, *May*, 1185

Hemolytic jaundice in thrombo-ulcerative endocarditis, *July*, 183
splenectomy in, *Sept.*, 416
Hemopneumothorax, *May*, 1159
diagnosis, *May*, 1162, 1164
exploratory puncture in, *May*, 1163
from trauma, *May*, 1166
from tumor, *May*, 1166
high-grade scoliosis and, *May*, 1172
history of case, *May*, 1159
physical examination, *May*, 1161, 1168
roentgen-ray examination in, *May*, 1171
Schrumpfung in, *May*, 1171
treatment, *May*, 1172
tuberculin test in, *May*, 1168
tuberculosis in, *May*, 1165
Woulff bottle exercises in, *May*, 1173
Hemoptysis in recurrent endocarditis, *Nov.*, 601
simulating hematemesis, *May*, 1284
history of case, *May*, 1284
Hemorrhage in carcinoma of rectum, *Jan.*, 739
in latent gastric ulcer, *Jan.*, 761
in thrombo-ulcerative endocarditis, *July*, 183
Hemorrhagic pericarditis, *March*, 918. See also *Pericarditis, hemorrhagic.*
Hemorrhoids from chronic constipation, *March*, 1080
in etiology of carcinoma of rectum, *Jan.*, 738
rectal stricture after, *Jan.*, 671
history of case, *Jan.*, 671
physical examination, *Jan.*, 671
treatment, *Jan.*, 672
Hepar lobatum in syphilitic cirrhosis of liver, *May*, 1279
multilobulatum in syphilis of liver, *Sept.*, 243
Hepatic flexure of colon, carcinoma of, 1237. See also *Carcinoma of hepatic flexure of colon.*
Hepatogenous jaundice, *May*, 1098
illustrative cases, *May*, 1098, 1101
Hering's ventricular stagnation pulse wave, *May*, 1202
Herzdruck in purulent pericarditis, *March*, 918
Hexamethylenamin in acute disseminated myelitis, *Jan.*, 726
Hirschfelder and Gibson's pulse wave, *May*, 1203
Hirschsprung's disease, relation of, to chronic constipation, *March*, 1086
His, bundle of, *May*, 1201
Hoarseness in aneurysm of aorta, *March*, 1025
Hodgkin's disease, mediastinal tumor similar to, *July*, 166
Hour-glass deformity in syphilis of stomach, *Sept.*, 363
Huchard's sign in aneurysm of aorta, *March*, 1019
Hunger-pains in achylia gastrica, *Jan.*, 698
in gastric ulcer, *Nov.*, 441, 442
Hydremic form of starch injury in nutrition of infants, *Jan.*, 801
Hydrochloric acid, free, as cause of gastric ulcer, *Nov.*, 441
Hydronephrosis, bilateral, polycystic kidneys and, differentiation, *Nov.*, 580
Hydropneumothorax in mediastinal tumor, *July*, 170
Hypersplenism, *Sept.*, 414
Hyperthyroidism, chlorosis and, differentiation, *March*, 975
in pulmonary tuberculosis, *Jan.*, 757
Hypertrophy of heart, *July*, 2
Hypogalactia, *July*, 123
Hypoplastic anemia, *May*, 1180

Icterus, *May*, 1091. See also *Jaundice.*
Immunity, breast feeding and, *July*, 118
Inanition fever of infants, *July*, 125
in Allen treatment of diabetes mellitus, *March*, 999
Infantile paralysis, *Nov.*, 463, 489
abortive form, *Nov.*, 480
acute onset, *Nov.*, 489
adrenalin by intraspinal injection in, *Nov.*, 486
ataxic type, *Nov.*, 479
blood examination in, *Nov.*, 476
bulbar type, *Nov.*, 479
cause, *Nov.*, 473
complicating syphilis, *Nov.*, 501, 502
diagnosis, *Nov.*, 482, 493, 496
differential, *Nov.*, 482, 502
disinfection of excretions in, *Nov.*, 484
dosage of serum in, *Nov.*, 487
electricity in, *Nov.*, 485, 507
encephalitic type, *Nov.*, 479
epidemic of, 1916, *Nov.*, 469
epidemiology, *Nov.*, 466
etiology, *Nov.*, 469
extent of muscle involvement in, *Nov.*, 506
factors in spread of, *Nov.*, 483
Flexner and Amoss' immune serum in, *Nov.*, 484
Flexner's serum treatment, *Nov.*, 507
hematogenous infection in, *Nov.*, 494

Infantile paralysis, historical study, *Nov.*, 465
histories of cases, *Nov.*, 463, 490, 494, 497, 501
incubation period, *Nov.*, 474
initial febrile period, *Nov.*, 474
involvement of central nervous system in, *Nov.*, 504
Landry's paralysis type, *Nov.*, 478
Levaditi's bodies in, *Nov.*, 471, 472
lumbar puncture in, *Nov.*, 507
massage in, *Nov.*, 485
meningeal type, *Nov.*, 479, 496
methods of spread, *Nov.*, 467
in body, *Nov.*, 473
micro-organisms in, *Nov.*, 470
mortality in, *Nov.*, 481
mouth disinfection in, *Nov.*, 484
muscle tests after, *Nov.*, 486
training after, *Nov.*, 486
training in, *Nov.*, 485
nervous phenomena, *Nov.*, 476
nose disinfection in, *Nov.*, 484
orthopedic treatment, *Nov.*, 485, 508
paralytic stage, *Nov.*, 477
passive exercises in, *Nov.*, 507
pathogenesis, *Nov.*, 482
pathology, *Nov.*, 503
physical examination, *Nov.*, 491, 492, 495, 498
plaster immobilization in, *Nov.*, 507
points of entrance, *Nov.*, 472
poliomyelitis type, *Nov.*, 477
polyneuritis type, *Nov.*, 479
pontine type, *Nov.*, 479
prodromal stage, *Nov.*, 474
prodromata, *Nov.*, 489
prognosis, *Nov.*, 481
prophylaxis, *Nov.*, 483
reparative stage, *Nov.*, 480
residual paralysis in, *Nov.*, 481
rest in, *Nov.*, 507
school attendance in spread of, *Nov.*, 483
serum treatment, *Nov.*, 484, 486, 507
skin in, *Nov.*, 476
spinal fluid in, *Nov.*, 482
support for limbs in, *Nov.*, 405
symptomatology, *Nov.*, 473
toe-drop in, *Nov.*, 485
treatment, *Nov.*, 483, 506
of pain in, *Nov.*, 485
types, *Nov.*, 477
vaccination in, *Nov.*, 484
virus of, peculiarities, *Nov.*, 471
viability in body, *Nov.*, 473
Wickmann's classification, *Nov.*, 506
Infants, alkalipenia in, *Jan.*, 800
Infants, atrophy in, *Jan.*, 805. See also *Decomposition.*
breast-fed, resistance of, to disease, *Jan.*, 796
cachectic purpura in, *Jan.*, 807
decomposition in, *Jan.*, 805. See also *Decomposition.*
exudative diathesis in, *Jan.*, 797
fat-feeding of, relation to resistance and nutritional disturbances, *Jan.*, 799
inanition fever of, *July*, 125
marantic purpura in, *Jan.*, 807
marasmus in, *Jan.*, 805. See also *Decomposition.*
normal, artificial feeding of. See *Artificial feeding.*
condition of monothermia in, *July*, 113
energy requirement of, *July*, 124
feeding of. See *Breast feeding.*
muscle tonus in, *July*, 114
turgor in, *July*, 113
nutrition of, disease resistance in relation to, *Jan.*, 795
mineral salts in relation to, *Jan.*, 802
starch injury in, *Jan.*, 800
pyelitis in, *March*, 895. See also *Pyelitis in infancy.*
resistance of, to thrush, *Jan.*, 797
septic infections in, *July*, 126
sick, advantages of breast milk for, *July*, 128
so-called vomiting in, *July*, 57
underfeeding of, results, *Jan.*, 80
Infarct in spleen in recurrent endocarditis, *Nov.*, 600
pulmonary, in duodenal ulcer, *Jan.*, 773
Influenza in etiology of pulmonary tuberculosis, *Jan.*, 749
Insane, general paralysis of, paralysis agitans and, differentiation, *March*, 958
Insanity, manic-depressive, *July*, 75
diagnosis, *July*, 77
dysthyroidism as cause, *July*, 78
history of case, *July*, 75
treatment, *July*, 78
Insomnia, digitalis in, *July*, 11
Insufficiency, cardiac, digitalis in, *July*, 5
mitral, digitalis in, *July*, 5
Intermittent claudication, *July*, 187
blood examination in, *July*, 18
history, *July*, 187
physical examination, *July*, 187
prognosis, *July*, 191
treatment, *July*, 191
pulse beat, *May*, 1207

Intestinal flatulence, relation of chronic constipation to, *March*, 1083
retroperitoneal sarcoma and, differentiation, *March*, 1047
Intestines, Bacillus aërogenes capsulatus infection of, *March*, 1007. See also *Bacillus aërogenes capsulatus infection of intestines*.
Involution, catatonia of, *Jan.*, 714
Iodid acne, *Sept.*, 319
Ipecac in amebic dysentery, *Jan.*, 680
Iron in chlorosis, *March*, 984
subcutaneous injection, *March*, 987
in pernicious anemia, *Nov.*, 532

JAUNDICE, *May*, 1091
catarrhal, illustrative case, *May*, 1002
classification of, from pathologic standpoint, *May*, 1097
from physiologic standpoint, *May*, 1098
dissociation, in pernicious anemia, illustrative case, *May*, 1106
hematogenous, illustrative case, *May*, 1104
hemolytic, splenectomy in, *Sept.*, 416
hepatogenous, *May*, 1098
illustrative cases, *May*, 1098, 1101
historical notes, *May*, 1092
in carcinoma of head of pancreas, *Sept.*, 303
in syphilis of liver, *Sept.*, 247
obstructive, *May*, 1092
physiology of liver in relation to, *May*, 1095
Juvenile arhythmia, *May*, 1024

KARELL diet, digitalis with, *July*, 6
Keith and Flack's node, *May*, 1200
Keller's malt soup in artificial feeding, *Sept.*, 268
Kernig sign in cerebrospinal meningitis, *July*, 195
Kidney disease, vomiting from, *July*, 59
displaced, with splanchnoptosis, *Jan.*, 635
infections from chronic constipation, *March*, 1084
involvement in splanchnoptosis, *Jan.*, 641
polycystic, *Nov.*, 577
test, two-hour, *March*, 1029
Koch's old tuberculin in diagnosis of incipient pulmonary tuberculosis, 754
Krönig's apical percussion in pulmonary tuberculosis, *March*, 966

LABORATORY findings in rheumatoid arthritis, *March*, 870
Lamblia intestinalis in protozoic enterocolitis, *May*, 1134
Landry's paralysis type of infantile paralysis, *Nov.*, 478
Lane's kinks in chronic constipation, *March*, 1078
Larosan milk in infant feeding, *Sept.*, 268
Lead-colic in pleurisy with gastric spasm, *Sept.*, 377
Lead-poisoning, wrist-drop in, *Sept.*, 378
Leaded gloves for operator in fluoroscopy, *July*, 206
Leube and Arneth's leukanemia, *May*, 1179
Leukanemia of Leube and Arneth, *May*, 1179
Leukemia, acute lymphatic, purpura hæmorrhagica and, differentiation, *Jan.*, 859
myelogenous, *Sept.*, 385. See also *Myelogenous leukemia*.
skin pigmentation in, *Nov.*, 550
Leukocyte count in pernicious anemia, *Nov.*, 511
Leukopenia in miliary tuberculosis, *Sept.*, 231
Levaditi's bodies in infantile paralysis, *Nov.*, 471, 472
Littré's glands, involvement of, in specific urethritis, *March*, 1062
Liver, cirrhosis of, caput medusæ in, *May*, 1251
retroperitoneal sarcoma and, differentiation, *March*, 1045
syphilis of liver and, differentiation, *Sept.*, 242
syphilitic, *May*, 1278. See also *Syphilitic cirrhosis of liver*.
physiology of, in relation to jaundice, *May*, 1095
syphilis of, *Sept.*, 237
Locomotor ataxia, *May*, 1217. See also *Tabes dorsalis*.
Lumbar puncture in acute disseminated myelitis, *Jan.*, 723
syphilitic meningomyelitis, *Jan.*, 727
in infantile paralysis, *Nov.*, 507
in pernicious anemia, *Nov.*, 525
in progressive muscular atrophy, *Sept.*, 295
Lung abscess after croupous pneumonia, *Jan.*, 661. See also *Pulmonary abscess*.
fibroid induration of, *May*, 1261
gangrene of, *Nov.*, 557
syphilitic infection of, *May*, 1253, 1261. See also *Syphilitic infection of lungs*.
Lymphangitis, breast feeding in, *July*, 120

Mackenzie's nodal rhythm, *May*, 1208
ventricular pulse wave, *May*, 1202
Magenblase in fluoroscopy, *July*, 209
Malaria, chronic, pernicious anemia and, differentiation, *May*, 1184
Manganese in pernicious anemia, *Nov.*, 532
Mange-cure dermatitis, *Nov.*, 540
Manic-depressive insanity, *July*, 75
diagnosis, *July*, 77
dysthyroidism as cause, *July*, 78
history of case, *July*, 75
treatment, *July*, 78
Marantic purpura in infants, *Jan.*, 807
Marasmus in infants, *Jan.*, 805. See also *Decomposition.*
Marriott's method of determining reserve alkalinity in acidosis, *Jan.*, 684
of estimating acidosis, *Jan.*, 684
Massage, facial, in treatment of acne, *Sept.*, 323
in chronic constipation, *March*, 1090
in infantile paralysis, *Nov.*, 485
Mastitis, breast feeding in, *July*, 120
Masturbation, mental effects of, *July*, 68
Maternal breasts, varieties, *July*, 116
Mediastinal tumor, *July*, 163
diagnosis, *July*, 166
gangrene of lung from, *Nov.*, 562
history of case, *July*, 163
hydropneumothorax in, *July*, 170
physical examination, *July*, 165
pneumothorax in, *July*, 168
prognosis, *July*, 167
similarity to Hodgkin's disease, *July*, 166
Medin's classification of infantile paralysis, *Nov.*, 477
Megacolon, relation of, to chronic constipation, *March*, 1086
Megalocytes in pernicious anemia, *Nov.*, 511
Megalomania in general paresis, *July*, 72
Melancholia, recurrent, *July*, 75
diagnosis, *July*, 77
dysthyroidism as cause, *July*, 78
history of case, *July*, 75
treatment, *July*, 78
Melanoderma in case presenting Addison's syndrome, *Nov.*, 549
Meningeal type of infantile paralysis, *Nov.*, 479, 496
Meningitis, cerebrospinal, *July*, 193
diagnosis, *July*, 196
Flexner's serum in, *July*, 197
history of case, *July*, 193
Kernig sign in, *July*, 195
physical examination, *July*, 194
Meningitis cerebrospinal, treatment, *July*, 197
tuberculous, acute syphilitic meningomyelitis and, differentiation, *Jan.*, 729
miliary tuberculosis and, differentiation, *Sept.*, 228
Meningomyelitis, syphilitic, acute, *Jan.*, 726
diagnosis, *Jan.*, 728
history of case, *Jan.*, 726
lumbar puncture in, *Jan.*, 727
physical examination, *Jan.*, 726
prognosis, *Jan.*, 729
reflexes in, *Jan.*, 727
treatment, *Jan.*, 729
tuberculous meningitis and, differentiation, *Jan.*, 729
Menstruation, effect of, on breast milk, *July*, 121
Mental disorders due to oral infections, *July*, 136
Middle West, protozoic enterocolitis in, *May*, 1109
Migraine, vomiting from, *July*, 61
Miliary tuberculosis, acute, *Sept.*, 221
absence of lung symptoms in, *Sept.*, 234
autopsy findings in, *Sept.*, 233
blood examination in, *Sept.*, 225
course, *Sept.*, 221
diagnosis, *Sept.*, 226
endocarditis and, differentiation, *Sept.*, 228
further report on, *Sept.*, 232
history of case, *Sept.*, 221
leukopenia in, *Sept.*, 231
onset, *Sept.*, 221
physical examination, *Sept.*, 222
temperature-curve in, *Sept.*, 223, 224
trichinosis and, differentiation, *Sept.*, 228
tubercular meningitis and, differentiation, *Sept.*, 228
typhoid fever and, differentiation, *Sept.*, 225, 228
urinalysis in, *Sept.*, 224
Milk, albumin, in decomposition in infants, *Jan.*, 815
breast. See *Breast milk.*
cows'. See *Cows' milk.*
certified. See *Certified milk.*
goats', for artificial feeding, *Sept.*, 256
Milk-borne diseases, *Sept.*, 256
Mineral salts in relation to nutrition of infants, *Jan.*, 802
Mitral insufficiency, digitalis in, *July*, 5
stenosis, digitalis in, *July*, 5
Monothermia in normal infant, *July*, 113

Morawitz's classification of anemias, *May*, 1178
theory of chlorosis, *March*, 982
Morbus maculosus, *Jan.*, 849. See also *Purpura hæmorrhagica*.
Morgagni's crypts, involvement of, in specific urethritis, *March*, 1062
Morphin habit in pleurisy with gastric spasm, *Sept.*, 380
with digitalis, *July*, 11
Mortality in infantile paralysis, *Nov.*, 481
Mosenthal diet in acute nephritis following tonsillitis, *May*, 1309
modification of Hedinger-Schlayer renal test, *March*, 1029
Motor aphasia, *July*, 103
Mouth infections, *July*, 133
Müller's theory of chlorosis, *March*, 982
Muscle involvement, extent of, in infantile paralysis, *Nov.*, 506
tests after infantile paralysis, *Nov.*, 486
training in infantile paralysis, *Nov.*, 485, 486
Muscular atrophy, progressive, *Sept.*, 289
antisyphilitic treatment, *Sept.*, 297
Charcot type and, differentiation, *Sept.*, 295
history of case, *Sept.*, 289
lumbar puncture in, *Sept.*, 295
physical examination, *Sept.*, 290
syphilis in etiology, *Sept.*, 297
treatment, *Sept.*, 297
Myelin sheaths, destruction of, in multiple sclerosis, *Sept.*, 272
Myelitis, acute disseminated, *Jan.*, 721
diagnosis, *Jan.*, 724
history of case, *Jan.*, 721
lumbar puncture in, *Jan.*, 723
multiple sclerosis and, differentiation, *Jan.*, 725
physical examination, *Jan.*, 722
reflexes in, *Jan.*, 722
syphilis and, differentiation, *Jan.*, 725
treatment, *Jan.*, 726
Myelogenous leukemia, *Sept.*, 385
appendicitis and, differentiation, *Sept.*, 388
benzin in, *Sept.*, 393
blood examination in, *Sept.*, 391
diagnosis, *Sept.*, 392
duodenal ulcer and, differentiation, *Sept.*, 388
epistaxis in, *Sept.*, 391
gastric ulcer and, differentiation, *Sept.*, 387
history of case, *Sept.*, 385
Myelogenous leukemia, intermittent fever in, *Sept.*, 389
pernicious anemia and, differentiation, *Sept.*, 404
physical examination, *Sept.*, 390
symptoms, *Sept.*, 389
treatment, *Sept.*, 392
Myelopathic anemia, *May*, 1179
Myocarditis with diabetes mellitus, *July*, 23

Naegeli's grouping of chlorosis, *March*, 979
Nausea, rôle of, in vomiting, *July*, 61
Neosalvarsan in pernicious anemia, *Nov.*, 519
Nephritis, acute, following tonsillitis, *May*, 1307
history of case, *May*, 1307
physical examination, *May*, 1308
treatment, *May*, 1309
chronic, in atypical gout, *Nov.*, 605
interstitial, in diabetes mellitus, *July*, 13
in etiology of serofibrinous pericarditis, *March*, 934
Nervous system, central, causes of vomiting acting from, *July*, 60
involvement of, in infantile paralysis, *Nov.*, 504
depression of, chronic constipation from, *March*, 1075
vomiting, treatment, *July*, 62
Neuralgia in staphylococcic osteomyelitis, cause of, *July*, 94
phrenic nerve, in duodenal ulcer, *Jan.*, 782
Neurasthenia at onset of general paresis, *July*, 70
attitudinizing in, *Nov.*, 715
catatonia of involution in, *Jan.*, 714
histories of cases, *Jan.*, 707, 712, 717
in chronic constipation, treatment, *March*, 1090
trances in, *Jan.*, 712, 713
Neuritis, chronic peripheral, in diabetes mellitus, *July*, 13, 23
multiple, tabes dorsalis and, differentiation, *May*, 1232
peripheral, *July*, 81
diagnosis, *July*, 82
history of case, *July*, 81
physical examination, *July*, 81
reflexes in, *July*, 82
sensory disturbances in, *July*, 81
treatment, antisyphilitic, *July*, 83
sciatic, in duodenal ulcer, *Jan.*, 780
Neuropathic diathesis in infants, *July*, 128

Node, auriculoventricular, *May*, 1201
Keith and Flack's, *May*, 1201
sino-auricular, *May*, 1200
Tawara's, *May*, 1201

Occupational dermatitis, acquired susceptibility in, *Nov.*, 543
treatment, *Nov.*, 544
Oligocythemia in pernicious anemia, *Sept.*, 397
Opaque enema, fluoroscopic examination after injection, *Jan.*, 834
technic of injection, *Jan.*, 832
meal, time for passage of, through gastro-intestinal tract, *Jan.*, 828
Open pneumothorax, *May*, 1298
Ophthalmoplegia, external, *Sept.*, 279
antisyphilitic treatment, *Sept.*, 287
history of case, *Sept.*, 279
physical examination, *Sept.*, 279
reflexes in, *Sept.*, 280
strabismus in, *Sept.*, 283
symptoms, *Sept.*, 285
treatment, *Sept.*, 287
Optic nerve atrophy, primary, tabes dorsalis and, differentiation, *May*, 1232
Oral bacteria, varieties, *July*, 133
foci of infection, classification, *March*, 992
low-grade secondary infection, *March*, 996
relation of, to systemic diseases, *March*, 991
infections, *July*, 133
action of autogenous vaccines in, *March*, 992
active treatment, *July*, 142
avenues for escape of pus in, *July*, 134
chief sources of, *July*, 134
classification of possible results of, 137
contra-indications to removal of teeth in, *July*, 147
diagnosis of, by general practitioner, *July*, 139
effects of ingested pus in, *July*, 135
on general nutrition, *July*, 138
in etiology of disease, *July*, 135
mental disorders due to, *July*, 136
mild, local effects, *July*, 134
prophylaxis, *July*, 141
radiography of teeth in, *July*, 143
treatment, *July*, 141
Orthoform dermatitis, cases of, *Nov.*, 535
Orthopedic treatment of infantile paralysis, *Nov.*, 485, 508
Orthopercussion, Goldscheider's, in pulmonary tuberculosis, *March*, 966
Osler's classification of gonorrheal arthritis, *May*, 1191
Osteomyelitis, staphylococcic, *July*, 89
diagnosis, *July*, 93
history of case, *July*, 89
infected teeth in etiology, *July*, 91
neuralgia in, cause of, *July*, 94
operations in, *July*, 91, 93

Pain, absence of, in cardiospasm, *Nov.*, 620
digitalis in, *July*, 12
epigastric, in achylia gastrica, *Jan.*, 698
in carcinoma of rectum, *Jan.*, 739
in gastric ulcer, explanation, *Nov.*, 442
Palpation in early pulmonary tuberculosis, *Jan.*, 746
Palpitation, digitalis in, *July*, 12
Palsy, shaking, *March*, 949. See also *Paralysis agitans.*
Pancreas, carcinoma of head, *Sept.*, 303
diseases of, skin pigmentation in, *Nov.*, 550
extirpation of, diabetes mellitus from, *March*, 1000
Papillomata of bladder, *March*, 941
cystoscopic examinations, *March*, 942, 943
fulguration treatment, *March*, 942, 943
local reaction, *March*, 948
number required, *March*, 947
pain in, *March*, 946
results from, *March*, 947
selection of cases for, *March*, 945
technic, *March*, 944
where to begin, *March*, 946
hematuria in, *March*, 942, 943
history of case, *March*, 941
importance of early diagnosis, *March*, 943
open treatment, *March*, 944
recurring, treatment, *March*, 946
Paracentesis abdominis for ascites in retroperitoneal sarcoma, *March*, 1047
in carcinoma of hepatic flexure of colon, *May*, 1242
Paralysis agitans, *March*, 949
brain changes in, *March*, 950
drug treatment, *March*, 961
etiology, *March*, 949
general paralysis of insane and, differentiation, *March*, 958
histories of cases, *March*, 952, 953, 954
multiple sclerosis and, differentiation, *March*, 957

Paralysis agitans, physical examination, *March*, 952, 953, 954
pseudobulbar paralysis and, differentiation, *March*, 960
symptoms in, *March*, 951
treatment, *March*, 961
variation of symptoms, *March*, 959
Wilson's disease and, differentiation, *March*, 951, 958
infantile. See *Infantile paralysis*.
of insane, paralysis agitans and, differentiation, *March*, 958
pseudobulbar, paralysis agitans and, differentiation, *March*, 960
Paraphasia, *July*, 97
apraxia in, *July*, 99
blood examination in, *July*, 98
conclusions on, *July*, 109
history of case, *July*, 98
thrombosis in etiology, *July*, 102, 109
treatment, *July*, 111
Wernicke's conduction, *July*, 103
Paraphenylendiamin dermatitis, cases of, *Nov.*, 538, 539
Paraplegia in pernicious anemia, *Nov.*, 523
spastic spinal, in pernicious anemia, *Nov.*, 516
Parenteral infections in infants, breast feeding in, *July*, 120
Paresis, general, *July*, 65
antisyphilitic treatment, *July*, 73
cerebrospinal fluid in, *July*, 67
early, *Sept.*, 299
antisyphilitic treatment, *Sept.*, 301
history of case, *Sept.*, 299
physical examination, *Sept.*, 299
syphilis in etiology, *Sept.*, 301
treatment, *Sept.*, 301
history of case, *July*, 67
megalomania in, *July*, 72
mental symptoms, *July*, 71
methods of onset, *July*, 71
neurasthenia at onset of, *July*, 71
physical examination, *July*, 65
sensory disturbances in, *July*, 66
syphilis in etiology, *July*, 71
treatment, antisyphilitic, *July*, 73
Parkinson's disease, *March*, 949. See also *Paralysis agitans*.
Paroxysmal tachycardia, *May*, 1214. See also *Tachycardia, paroxysmal*.
Passive exercises in infantile paralysis, *Nov.*, 507
Pegnin milk in artificial feeding, *Sept.*, 269
Pellagra, skin pigmentation from, *Nov.*, 550
Pelvic diseases, female, vomiting from, *July*, 60
skin pigmentation from, *Nov.*, 550
Percentage system of artificial feeding, *Sept.*, 261
Percussion in early pulmonary tuberculosis, *Jan.*, 747
Krönig's apical, in pulmonary tuberculosis, *March*, 966
threshold, in pulmonary tuberculosis, *March*, 966
Pericardial friction-rub in seropurulent pericarditis, *March*, 929
Pericardiomediastinitis, *Jan.*, 659
diagnosis, *Jan.*, 658
exploratory pericardial puncture, *Jan.*, 659
history of case, *Jan.*, 657
physical examination, *Jan.*, 657
secondary to acute plastic pericarditis, *Jan.*, 659
Pericarditis, acute plastic, pericardiomediastinitis secondary to, *Jan.*, 659
hemorrhagic, *March*, 918
appearance of skin, *March*, 921
diagnosis, *March*, 922
endocarditis in, *March*, 924
heart examination, *March*, 919
history of case, *March*, 918
physical examination, *March*, 919
purulent, *March*, 907
aspiration of pericardium in, *March*, 912
diagnosis, *March*, 911
etiology, *March*, 912
heart findings in, *March*, 910
herzdruck in, *March*, 918
history of case, *March*, 907
pericardotomy in, *March*, 915
physical examination, *March*, 908
postmortem findings, *March*, 916, 925
postoperative symptoms, *March*, 915
postural treatment, *March*, 917
pre-operative symptoms, *March*, 915
prognosis, *March*, 918
treatment, *March*, 913
surgical, *March*, 915, 917
serofibrinous, *March*, 927
apex-beat in, *March*, 931
aspiration in, *March*, 937
chronic interstitial nephritis in etiology, *March*, 934
diagnosis, *March*, 929
endocardial murmur in, *March*, 931
history of case, *March*, 927
indications for exploratory puncture in, *March*, 935
nature of exudate in, *March*, 934

Pericarditis, ventricular pain in, *March*, 930
pericardial friction-rub in, *March*, 929
physical examination, *March*, 928
pneumonia in etiology, *March*, 934
relative positions of heart and exudate in, *March*, 927
tuberculin in etiology, *March*, 934
treatment, *March*, 935
Pericardium, aspiration of, in purulent pericarditis, *March*, 932
Pericardiotomy in purulent pericarditis, *March*, 935
Peripheral neuritis, *July*, 82
diagnosis, *July*, 82
history of case, *July*, 81
physical examination, *July*, 82
reflexes in, *July*, 82
sensory disturbances in, *July*, 82
treatment, antisyphilitic, *July*, 83
Peristalsis, intestinal, factors affecting, *March*, 1015
waves of, in fluoroscopy of stomach, *July*, 212
Peritonitis, fibrous, associated with syphilis, retroperitoneal sarcoma and, differentiation, *March*, 1041
neoplastic, retroperitoneal sarcoma and, differentiation, *March*, 1051
Pernicious anemia, *Sept.*, 397; *May*, 1175
anemia leukemicum infantum and, differentiation, *Sept.*, 405
anisocytosis in, *Sept.*, 397
antisyphilitic treatment, *Nov.*, 519, 1186
aplastic, *Nov.*, 512
arsenic in, *Nov.*, 519
astereognosis in, *Nov.*, 520
ataxic paraplegic type, *Nov.*, 515
blood crises in, *Sept.*, 402
examination in, *Sept.*, 409; *May*, 1176
findings in, *Nov.*, 512
blood-platelets in, *Sept.*, 401
carcinomatosis of bone-marrow and, differentiation, *Sept.*, 405; *May*, 1184
carcinoma of stomach and, differentiation, *May*, 1184
chronic malaria and, differentiation, *May*, 1184
diagnosis, *Sept.*, 410; *May*, 1181, 1185
differential, *May*, 1182
diarsenol in, *Nov.*, 531
differential diagnosis, *Sept.*, 395
dissociation jaundice in, history of case, *May*, 1106
fever in, *Nov.*, 518
Pernicious anemia, gastric sensations in, *Nov.*, 513
hemicrania in, *May*, 1181. See also *Hemicrania in pernicious anemia*
history of case, *Sept.*, 406; *May*, 1180
iron in, *Nov.*, 521
laboratory findings, *Sept.*, 408
leukocyte count in, *Nov.*, 511
low-grade fever in, *Sept.*, 407
lumbar puncture in, *Nov.*, 515
manganese in, *Nov.*, 521
megaloblasts in, *Nov.*, 511
myelogenous leukemia and, differentiation, *Sept.*, 404
nature of, *Nov.*, 517
neurohumors in, *Nov.*, 519
nervous complications, *Nov.*, 513
oligocythemia in, *Sept.*, 397
paraplegia in, *Nov.*, 515
physical examination, *Sept.*, 407; *Nov.*, 512, 518; *May*, 1176
poikilocytosis in, *Sept.*, 398
polychromasia in, *Sept.*, 398
posterolateral cord involvement in, *Nov.*, 517
primary, *Nov.*, 509
color-index of blood in, *Nov.*, 509
histories of cases, *Nov.*, 500, 530
overproduction of hemoglobin in, *Nov.*, 510
prognosis, *Nov.*, 530
pyknosis in, *Sept.*, 399
red blood-corpuscles in, *Sept.*, 401
remissions in, *Sept.*, 410; *Nov.*, 519
results of treatment, *May*, 1186
significance of megaloblast in, *Sept.*, 403
sodium cacodylate in, *Nov.*, 519
spastic spinal paraplegia in, *Nov.*, 516
spinal cord involvement in, *Nov.*, 513
splenectomy in, *Sept.*, 414
splenic enlargement in, *Nov.*, 518
syphilis and, analogy between, *Nov.*, 519
tabetic type, *Nov.*, 513
theory of infectious origin, *Nov.*, 517
treatment, *Nov.*, 519, 526, 531; *May*, 1186
results of, *Nov.*, 532
variations in blood-picture, *Sept.*, 410
von Jaksch's anemia and, differentiation, *Sept.*, 405
white blood-corpuscles in, *Sept.*, 401
Pettenkofer test for bile acids and salts, *May*, 1095

Philippines, protozoic enterocolitis in, *May*, 1130
Phrenic nerve neuralgia in duodenal ulcer, *Mon.*, 782
Phthalein output, absence of, in polycystic kidney, *Mon.*, 585
Pigment dermatitis, *Mon.*, 542
Pleura, disease of, gangrene of lung from, *Mon.*, 502
Pleurisy, *Sept.*, 375
  gastric spasm in, *Sept.*, 377
    morphin habit in, *Sept.*, 380
    treatment, *Sept.*, 381
    vomiting in, *Sept.*, 380
  history of case, *Sept.*, 375
  lead-colic in, *Sept.*, 377
Plummer's apparatus for dilation in cardiospasm, *Mon.*, 709
  hydrostatic guided bougie in cardiospasm, *Mon.*, 708
  perforated esophageal bougies in cardiospasm, *Mon.*, 708, 708
Pneumonia aspiration, gangrene of lung from, *Mon.*, 502
  croupous, gangrene of lung from, *Mon.*, 502
    pulmonary abscess after, *Jan.*, 601
    See also *Pulmonary abscess*.
  in etiology of serofibrinous pericarditis, *Monk*, 914
  lobar, gangrene of lung and, differentiation, *Mon.*, 500
  syphilitic, *May*, 1082
Pneumothorax, closed, *May*, 1297
  etiology, *May*, 1297
  in mediastinal tumor, *July*, 168
  open, *May*, 1298.
    treatment, *May*, 1299
  pulmonary tuberculosis in etiology, *May*, 1296
  valvular, *May*, 1288
    displacement of organs in, *May*, 1298
    due to emphysema, *May*, 1291
      history of case, *May*, 1291
      operation, *May*, 1304
      physical examination, *May*, 1292
      postmortem examination of lung in, *May*, 1296
      roentgen-ray examination in, *May*, 1293
      surgical treatment, *May*, 1296
      thoracentesis in, *May*, 1296
    prognosis, *May*, 1304
    Unverricht procedure in, *May*, 1294
    valvular drainage in, *May*, 1295
  varieties, *May*, 1297
Poikilocytosis in pernicious anemia, *Sept.*, 398
Poliomyelitis, acute anterior. See *Infantile paralysis*.
  type of infantile paralysis, *Mon.*, 477
Polychromasia in pernicious anemia, *Sept.*, 398
Polycystic kidneys, *Mon.*, 577
  absence of phthalein output in, *Mon.*, 585
  bilateral hydronephrosis and, differentiation, *Mon.*, 580
  clinical characteristics, *Mon.*, 580
  determining functional capacity in, *Mon.*, 584
  histories of cases, *Mon.*, 577, 582
  malignant neoplasm and, differentiation, *Mon.*, 579
  nature of disease, *Mon.*, 580
  physical examination, *Mon.*, 578, 582
  postmortem reports, *Mon.*, 588
  prognosis, *Mon.*, 580
  treatment, *Mon.*, 588
Polymeritic form of infantile paralysis, *Mon.*, 479
Pontine type of infantile paralysis, *Mon.*, 479
Pregnancy in diabetes mellitus, *July*, 47
    Cesarean section in, *July*, 49
    dietetic treatment, *July*, 49
    history of case, *July*, 47
  in etiology of colon bacillus pyelitis, *Mon.*, 568
  in renal diabetes, *July*, 50
  vomiting in, *July*, 60
Pressure pains in aneurysm of aorta, *Monk*, 1024
Primrose dermatitis, cases of, *Mon.*, 533, 534
Prostate, abscess of, in gonorrheal arthritis, *May*, 1161
  hypertrophy of, vomiting from, *July*, 60
Protein of cows' milk in artificial feeding, *Sept.*, 395
Protozoic carriers, *May*, 1110
Protozoic enterocolitis, *May*, 1109
    abdominal inspection, *May*, 1118
    symptoms in, *May*, 1120
    after-treatment, *May*, 1142
    age in etiology, *May*, 1121
    anemia in, *May*, 1107
    cercomonads in, *May*, 1114, 1118
    Cercomonas intestinalis hominis in, *May*, 1133
    character of diarrhea in, *May*, 1125
    clinical symptomatology, *May*, 1124
    constipation in, *May*, 1125
    contaminated vegetables in etiology, *May*, 1123
    diarrhea in, *May*, 1124
    duration of diarrhea in, *May*, 1125

Pericarditis, serofibrinous, pain in, *March*, 930
pericardial friction-rub in, *March*, 929
physical examination, *March*, 928
pneumonia in etiology, *March*, 934
relative positions of heart and exudate in, *March*, 937
tonsillitis in etiology, *March*, 934
treatment, *March*, 935
Pericardium, aspiration of, in purulent pericarditis, *March*, 912
Pericardotomy in purulent pericarditis, *March*, 915
Peripheral neuritis, *July*, 81
diagnosis, *July*, 82
history of case, *July*, 81
physical examination, *July*, 81
reflexes in, *July*, 82
sensory disturbances in, *July*, 81
treatment, antisyphilitic, *July*, 83
Peristalsis, intestinal, factors affecting, *March*, 1072
waves of, in fluoroscopy of stomach, *July*, 212
Peritonitis, fibrous, associated with syphilis, retroperitoneal sarcoma and, differentiation, *March*, 1047
neoplastic, retroperitoneal sarcoma and, differentiation, *March*, 1051
Pernicious anemia, *Sept.*, 395; *May*, 1175
anemia leukemicum infantum and, differentiation, *Sept.*, 405
anisocytosis in, *Sept.*, 397
antisyphilitic treatment, *Nov.*, 519, 1186
aplastic, *Nov.*, 512
arsenic in, *Nov.*, 519
astereognosis in, *Nov.*, 520
ataxic paraplegic type, *Nov.*, 515
blood crises in, *Sept.*, 402
examination in, *Sept.*, 409; *May*, 1176
findings in, *Nov.*, 512
blood-platelets in, *Sept.*, 401
carcinomatosis of bone-marrow and, differentiation, *Sept.*, 405; *May*, 1184
carcinoma of stomach and, differentiation, *May*, 1184
chronic malaria and, differentiation, *May*, 1184
diagnosis, *Sept.*, 410; *May*, 1181, 1185
differential, *May*, 1182
diarsenol in, *Nov.*, 531
differential diagnosis, *Sept.*, 395
dissociation jaundice in, history of case, *May*, 1106
fever in, *Nov.*, 518
Pernicious anemia, girdle sensations in, *Nov.*, 521
hematemesis in, *May*, 1286. See also *Hematemesis in pernicious anemia.*
history of case, *Sept.*, 406; *May*, 1186
iron in, *Nov.*, 532
laboratory findings, *Sept.*, 408
leukocyte count in, *Nov.*, 511
low-grade fever in, *Sept.*, 407
lumbar puncture in, *Nov.*, 525
manganese in, *Nov.*, 532
megalocytes in, *Nov.*, 511
myelogenous leukemia and, differentiation, *Sept.*, 404
nature of, *Nov.*, 517
neosalvarsan in, *Nov.*, 519
nervous complications, *Nov.*, 513
oligocythemia in, *Sept.*, 397
paraplegia in, *Nov.*, 523
physical examination, *Sept.*, 407; *Nov.*, 524, 528; *May*, 1176
poikilocytosis in, *Sept.*, 398
polychromasia in, *Sept.*, 398
posterolateral cord involvement in, *Nov.*, 517
primary, *Nov.*, 509
color-index of blood in, *Nov.*, 509
histories of cases, *Nov.*, 509, 520
overproduction of hemoglobin in, *Nov.*, 510
prognosis, *Nov.*, 520
pyknosis in, *Sept.*, 399
red blood-corpuscles in, *Sept.*, 401
remissions in, *Sept.*, 410; *Nov.*, 519
results of treatment, *May*, 1186
significance of megaloblast in, *Sept.*, 403
sodium cacodylate in, *Nov.*, 519
spastic spinal paraplegia in, *Nov.*, 516
spinal cord involvement in, *Nov.*, 513
splenectomy in, *Sept.*, 414
splenic enlargement in, *Nov.*, 518
syphilis and, analogy between, *Nov.*, 519
tabetic type, *Nov.*, 513
theory of infectious origin, *Nov.*, 517
treatment, *Nov.*, 519, 526, 531; *May*, 1186
results of, *Nov.*, 532
variations in blood-picture, *Sept.*, 410
von Jaksch's anemia and, differentiation, *Sept.*, 405
white blood-corpuscles in, *Sept.*, 401
Pettenkofer test for bile acids and salts, *May*, 1095

Philippines, protozoic enterocolitis in, *May*, 1131
Phrenic nerve neuralgia in duodenal ulcer, *Jan.*, 782
Phthalein output, absence of, in polycystic kidney, *Nov.*, 585
Pigment dermatitis, *Nov.*, 541
Pleura, disease of, gangrene of lung from, *Nov.*, 562
Pleurisy, *Sept.*, 375
  gastric spasm in, *Sept.*, 377
    morphin habit in, *Sept.*, 380
    treatment, *Sept.*, 381
    vomiting in, *Sept.*, 380
  history of case, *Sept.*, 375
  lead-colic in, *Sept.*, 377
Plummer's apparatus for dilation in cardiospasm, *Nov.*, 629
  hydrostatic guided bougie in cardiospasm, *Nov.*, 628
  perforated esophageal bougies in cardiospasm, *Nov.*, 626, 628
Pneumonia aspiration, gangrene of lung from, *Nov.*, 562
  croupous, gangrene of lung from, *Nov.*, 562
    pulmonary abscess after, *Jan.*, 661. See also *Pulmonary abscess.*
  in etiology of serofibrinous pericarditis, *March*, 934
  lobar, gangrene of lung and, differentiation, *Nov.*, 560
  syphilitic, *May*, 1261
Pneumothorax, closed, *May*, 1297
  etiology, *May*, 1297
  in mediastinal tumor, *July*, 168
  open, *May*, 1298.
    treatment, *May*, 1299
  pulmonary tuberculosis in etiology, *May*, 1296
  valvular, *May*, 1288
    displacement of organs in, *May*, 1298
    due to emphysema, *May*, 1291
      history of case, *May*, 1291
      operation, *May*, 1294
      physical examination, *May*, 1292
      postmortem examination of lung in, *May*, 1299
      roentgen-ray examination in, *May*, 1293
      surgical treatment, *May*, 1296
      thoracentesis in, *May*, 1292
    prognosis, *May*, 1304
    Unverricht procedure in, *May*, 1294
    valvular drainage in, *May*, 1295
  varieties, *May*, 1297
Poikilocytosis in pernicious anemia, *Sept.*, 398
Poliomyelitis, acute anterior. See *Infantile paralysis.*
  type of infantile paralysis, *Nov.*, 477
Polychromasia in pernicious anemia, *Sept.*, 398
Polycystic kidneys, *Nov.*, 577
  absence of phthalein output in, *Nov.*, 585
  bilateral hydronephrosis and, differentiation, *Nov.*, 580
  clinical characteristics, *Nov.*, 587
  determining functional capacity in, *Nov.*, 584
  histories of cases, *Nov.*, 577, 581
  malignant neoplasm and, differentiation, *Nov.*, 579
  nature of disease, *Nov.*, 586
  physical examination, *Nov.*, 578, 581
  postmortem reports, *Nov.*, 588
  prognosis, *Nov.*, 588
  treatment, *Nov.*, 588
Polyneuritis form of infantile paralysis, *Nov.*, 479
Pontine type of infantile paralysis, *Nov.*, 479
Pregnancy in diabetes mellitus, *July*, 47
    Cesarean section in, *July*, 49
    dietetic treatment, *July*, 49
    history of case, *July*, 47
  in etiology of colon bacillus pyelitis, *Nov.*, 568
  in renal diabetes, *July*, 50
  vomiting in, *July*, 60
Pressure pains in aneurysm of aorta, *March*, 1024
Primrose dermatitis, cases of, *Nov.*, 533, 534
Prostate, abscess of, in gonorrheal arthritis, *May*, 1191
  hypertrophy of, vomiting from, *July*, 60
Protein of cows' milk in artificial feeding, *Sept.*, 263
Protozoa carriers, *May*, 1110
Protozoic enterocolitis, *May*, 1109
  abdominal inspection, *May*, 1128
    symptoms in, *May*, 1126
  after-treatment, *May*, 1142
  age in etiology, *May*, 1121
  anemia in, *May*, 1127
  cercomonads in, *May*, 1114, 1118
  Cercomonas intestinalis hominis in, *May*, 1133
  character of diarrhea in, *May*, 1125
  clinical symptomatology, *May*, 1124
  constipation in, *May*, 1125
  contaminated vegetables in etiology, *May*, 1123
  diarrhea in, *May*, 1124
  duration of diarrhea in, *May*, 1125

Protozoic enterocolitis, dyspepsia, *May*, 1126
entameba cases, treatment, *May*, 1141
Entameba histolytica in, *May*, 1114
entamebæ in, description, *May*, 1133
etiologic factors, *May*, 1121
exacerbations in, *May*, 1110
examination of feces in, *May*, 1129, 1136, 1137
gastric analyses in, *May*, 1138
geographic distribution in etiology, *May*, 1122
histories of cases, *May*, 1111, 1115, 1119
in Middle West, *May*, 1109
in Philippines, *May*, 1131
laboratory examinations, *May*, 1113, 1117, 1129
Lamblia intestinalis in, *May*, 1134
measures to free intestine of protozoa in, *May*, 1140
mental attitude in, *May*, 1127
method of spread, *May*, 1011
nativity in etiology, *May*, 1122
palpation in, *May*, 1128
pancreatic efficiency tests in, *May*, 1137
physical examination, *May*, 1111, 1116, 1127
purpura hæmorrhagica and, differential, *Jan.*, 855
rectal examination in, *May*, 1128
strength loss in, *May*, 1127
test for bile-pigment in, *May*, 1138
testing for anemia in, *May*, 1139
thoracic examination, *May*, 1128
treatment, *May*, 1120, 1140
of entameba cases, *May*, 1141
of flagellate protozoa cases, *May*, 1142
trichomonads in, *May*, 1118
Trichomonas intestinalis hominis in, *May*, 1133
urinalysis in, *May*, 1140
varieties of protozoa in, *May*, 1109
water-supply in etiology, *May*, 1123
weight loss in, *May*, 1127
Pseudobulbar paralysis, paralysis agitans and, differentiation, *March*, 960
Pseudochlorosis of Müller, *March*, 983
Psychiatry, consideration of problems in, *Jan.*, 707
Psychic achylia, *Jan.*, 700
Psychotherapy in achylia gastrica, *Jan.*, 704
Pulmonary abscess after croupous pneumonia, *Jan.*, 661
diagnosis, *Jan.*, 664
further report, *Jan.*, 669
Pulmonary abscess after croupous pneumonia, history of case, *Jan.*, 661
physical examination, *Jan.*, 662
etiology, *Jan.*, 667
offensive sputum in, *Jan.*, 666
prognosis, *Jan.*, 668
surgical treatment, *Jan.*, 668
treatment, *Jan.*, 668
infarct in duodenal ulcer, *Jan.*, 773
tuberculosis, early, *Jan.*, 745. See also *Tuberculosis, early pulmonary.*
Pulse beat, absolutely irregular, *May*, 1208
alternating, *May*, 1211
dropped, *May*, 1207
intermittent, *May*, 1207
venous, newer analysis of, *May*, 1201
wave, auricular, *May*, 1202
carotid, *May*, 1202
Hering's ventricular stagnation, *May*, 1202
Hirschfelder and Gibson's, *May*, 1203
ventricular, of Mackenzie, *May*, 1202
Pulsus alternans, *May*, 1211
bigeminus, *May*, 1204, 1206
irregularis perpetuus, *May*, 1208
Puncture, exploratory, in hemopneumothorax, *May*, 1163
Purgatives in chronic constipation, *May*, 1088
Purpura hæmorrhagica, *Jan.*, 849
acute lymphatic leukemia and, differentiation, *Jan.*, 859
blood examination in, *Jan.*, 854
transfusion in, *Jan.*, 860
history of case, *Jan.*, 849
pernicious anemia and, differentiation, *Jan.*, 855
physical examination, *Jan.*, 850
scurvy and, differentiation, *Jan.*, 853
septic infection in etiology, *Jan.*, 852
treatment, *Jan.*, 860
marantic, in infants, *Jan.*, 807
Purulent pericarditis, *March*, 907. See *Pericarditis, purulent.*
Pus, avenues for escape of, in oral infections, *July*, 134
ingested, effects of, in oral infections, *July*, 135
Pyelitis from colon bacillus, *Nov.*, 567
associated with pathologic conditions, *Nov.*, 569
diagnosis, *Nov.*, 572
from acute infectious diseases, *Nov.*, 569

Pyelitis from colon bacillus from gastro-intestinal disturbances, *Nov.*, 569
from surgical operations, *Nov.*, 569
hematogenous infection in, *Nov.*, 570
internal medication in, *Nov.*, 575
lymphogenous infection in, *Nov.*, 571
matrimony in etiology, *Nov.*, 569
pelvic lavage in, *Nov.*, 573
predisposing factors, *Nov.*, 567
pregnancy in etiology, *Nov.*, 568
routes of infection in, *Nov.*, 570
sex in etiology, *Nov.*, 567
treatment, *Nov.*, 573
urogenous infection in, *Nov.*, 571
in infancy, *March*, 895
complications, *March*, 902
determining avenue of infection in, *March*, 898
diagnosis, *March*, 897
dietetic treatment, *March*, 903
etiology, *March*, 898
history of case, *March*, 895
modes of infection, *March*, 898, 899
physical examination, *March*, 896, 901
predisposing causes, *March*, 900
prognosis, *March*, 903
prophylaxis, *March*, 903
symptoms, *March*, 900
theories of, *March*, 898, 899
treatment, *March*, 903
alkalies in, *March*, 905
dietetic, *March*, 903
medicinal, *March*, 904
water in, *March*, 905
urinalysis in, *March*, 897, 900
urotropin in, *March*, 904
Pyknosis in pernicious anemia, *Sept.*, 399
Pylorus, stenosis of, cholelithiasis and, *Jan.*, 785
abdominal examination, *Jan.*, 787
blood examination in, *Jan.*, 787
diagnosis, *Jan.*, 787
history of case, *Jan.*, 785
operation in, *Jan.*, 792
pain in, *Jan.*, 789
physical examination, *Jan.*, 786
postoperative history, *Jan.*, 793
reflexes in, *Jan.*, 787
treatment, *Jan.*, 792
vomiting in, *Jan.*, 786, 790
Pyopericardium, *March*, 907. See also *Pericarditis, purulent.*
Radiography of teeth in oral infections, *July*, 143
Radiotherapy in carcinoma of esophagus, *May*, 1158
Radium treatment in cancer of rectum, *Jan.*, 743
Rectum, carcinoma of, *Jan.*, 731. See also *Carcinoma of rectum.*
stricture of, after hemorrhoids, *Jan.*, 671
history of case, 671
physical examination, *Jan.*, 671
treatment, *Jan.*, 672
Regurgitation, aortic, digitalis in, *July*, 5
in cardiospasm, *Nov.*, 620
of food in carcinoma of esophagus, *May*, 1151
vomiting and, differentiation, *July*, 56
Renal diabetes, pregnancy in, *July*, 50
disease, retroperitoneal sarcoma and, differentiation, *March*, 1045
glycosuria, *Sept.*, 339
history of case, *Sept.*, 339
treatment, *Sept.*, 340
test, two-hour, *March*, 1029
Reparation period in breast feeding, *July*, 129
Residual paralysis in infantile paralysis, *Nov.*, 481
Respiratory arhythmia, *May*, 1204
tract, syphilis of, *May*, 1260. See also *Syphilis of respiratory tract.*
Retching, *July*, 58
Retroperitoneal sarcoma, *March*, 1037
abdominal tumor in, *March*, 1049
localization of, *March*, 1050
actinotherapy in, *March*, 1055
ascites in, *March*, 1038
character of fluid, *March*, 1048, 1050
paracentesis for, *March*, 1047
cirrhosis of liver and, differentiation, *March*, 1045
Coley's serum in, *March*, 1055
fibrous peritonitis associated with syphilis and, differentiation, *March*, 1047
history of case, *March*, 1038
hospital course, *March*, 1041
intestinal obstruction and, differentiation, *March*, 1047
laboratory findings, *March*, 1041
neoplastic peritonitis and, differentiation, *March*, 1051
peritoneal tuberculosis and, differentiation, *March*, 1046
physical examination, *March*, 1038
postmortem findings in, *March*, 1056

Retroperitoneal sarcoma, preventing recurrence of ascites in, *March*, 1058
prognosis, *March*, 1053
recapitulation, *March*, 1043
renal disease and, differentiation, *March*, 1045
treatment, *March*, 1053
Rheumatism, articular, in etiology of serofibrinous pericarditis, *March*, 934
acute, atypical gout simulating, *Nov.*, 603, 604
Rheumatoid arthritis, anaphylaxis in, *March*, 869. See also *Anaphylaxis*.
Rhinophyma, *Sept.*, 319
Riegel's classification of conditions simulating achylia gastrica, *Jan.*, 704
Roentgenography by Cole's serial method, *July*, 218
fluoroscopy and, comparative value, *July*, 204
Roentgen-ray appearance of normal duodenal cap, *Nov.*, 433, 434
diagnosis of blind abscess of teeth, *March*, 995
examination, barium test-meal for, *Jan.*, 820
in abdominal aneurysm, *Jan.*, 676
in achylia gastrica, *Jan.*, 693, 695
in carcinoma of esophagus, *May*, 1153
of head of pancreas, *Sept.*, 305
of hepatic flexure of colon, *May*, 1243
in cardiospasm, *Nov.*, 621
in chronic bronchitis with emphysema, *Sept.*, 315
diarrhea, *Sept.*, 351
in early pulmonary tuberculosis, *Jan.*, 754
in gangrene of lung, *Nov.*, 565
in gastric ulcer, *Nov.*, 422
after medical treatment, *Nov.*, 425
value of, *Nov.*, 427, 438
in hemopneumothorax, *May*, 1171
in rheumatoid arthritis, *March*, 869
in splanchnoptosis, *Jan.*, 644
in syphilis of stomach, *Sept.*, 362, 368
in syphilitic infection of lungs, *May*, 1258
of gastro-intestinal tract, cleansing enemas before, *Jan.*, 821
in chlorosis, *March*, 974
laxatives before, *Jan.*, 845
opaque enema for, *Jan.*, 822
with barium, *Jan.*, 819
treatment in acne, *Sept.*, 324
Roentgen-ray treatment in cancer of rectum, *Jan.*,
Rumination, vomiting and, differentiation, *July*, 57

Saccharin water in artificial feeding, *Sept.*, 266
Salts, metabolism of, in artificial feeding, *Sept.*, 262
Salvarsan in syphilis of stomach, *Sept.*, 371
Sarcoma, retroperitoneal, *March*, 1037. See also *Retroperitoneal sarcoma*.
Schlesinger test for urobilin, *May*, 1094
Schrumpfung in hemopneumothorax, *May*, 1171
Sciatic neuritis in duodenal ulcer, *Jan.*, 780
Sclerosis, multiple, *Sept.*, 271
acute disseminated myelitis and, differentiation, *Jan.*, 725
destruction of myelin sheaths in, *Sept.*, 272
diagnosis, *Sept.*, 276
history of case, *Sept.*, 273
paralysis agitans and, differentiation, *March*, 957
pathology, *Sept.*, 271
physical examination, *Sept.*, 274
plaques in, *Sept.*, 272
scanning speech in, *Sept.*, 274
tonsillar infection in, *Sept.*, 277
Scoliosis, high-grade, hemopneumothorax and, *May*, 1172
Scurvy, purpura hæmorrhagica and, differentiation, *Jan.*, 853
Sea-sickness, vomiting from, *July*, 61
Selective vomiting, treatment, *July*, 62
Septic infections in infants, *July*, 126
Serofibrinous pericarditis, *March*, 927. See also *Pericarditis, serofibrinous*.
Serositis, multiple tubercular, *July*, 171
diagnosis, *July*, 172, 174
history of case, *July*, 171
physical examination, *July*, 171
Serum treatment of infantile paralysis, *Nov.*, 484, 486, 507
Sino-auricular node, *May*, 1200
Sinus arhythmia, *May*, 1204
Skin, pigmentation of, in Addison's disease, *Nov.*, 552
conditions producing, *Nov.*, 549
Sodium cacodylate in pernicious anemia, *Nov.*, 519
Spartein sulphate, action of, *July*, 9
Spasm at cardia in vagotonia, fluoroscopic examination, *Nov.*, 614
Speech tract, Wernicke's, *July*, 108

Spinal cord, combined degeneration of, tabes dorsalis and, differentiation, *May*, 1231
involvement in pernicious anemia, *Nov.*, 513
fluid examination in infantile paralysis, *Nov.*, 492
in infantile paralysis, *Nov.*, 482
Splanchnoptosis, *Jan.*, 631
characteristic abdomen in, *Jan.*, 633
chronic constipation in, *March*, 1084
colon in, *Jan.*, 642
constipation in, *Jan.*, 642
constitutional symptoms, *Jan.*, 643
diagnosis, *Jan.*, 643, 648, 650
dietetic treatment, *Jan.*, 645
displaced kidney with, *Jan.*, 635
dyspeptic disturbances in, *Jan.*, 641, 642
Einhorn's gastrodiaphane in, *Jan.*, 644
etiology, *Jan.*, 637
grouping, *Jan.*, 636
histories of cases, *Jan.*, 631, 647, 650
importance of after-care, *Jan.*, 651
in achylia gastrica, *Jan.*, 702
kidney involvement in, *Jan.*, 641
method of examination in, *Jan.*, 643
physical examination, *Jan.*, 633, 648
prognosis, *Jan.*, 644
roentgen rays in, *Jan.*, 644
stomach in, *Jan.*, 642
surgical treatment, *Jan.*, 649
symptomatology, *Jan.*, 640
treatment, *Jan.*, 644, 648, 651
dietetic, *Jan.*, 645
exercises in, *Jan.*, 646
rest, *Jan.*, 645
surgical, *Jan.*, 649
with diabetes mellitus, *July*, 73
Spleen, enlargement of, in pernicious anemia, *Nov.*, 518
in syphilis of liver, *Sept.*, 246
infarct in, in recurrent endocarditis, *Nov.*, 600
Splenectomy in Banti's disease, *Sept.*, 416
in Gaucher type of splenomegaly, *Sept.*, 416
in hemolytic jaundice, *Sept.*, 416
in pernicious anemia, *Sept.*, 414
in splenic anemia, *Sept.*, 416
in von Jaksch's anemia, *Sept.*, 416
Splenic anemia, splenectomy in, *Sept.*, 416
Splenomegaly, Gaucher type, splenectomy in, *Sept.*, 416
Sputum, offensive, in pulmonary abscess, *Jan.*, 666
in bronchiectasis, *July*, 177
Staphylococcic osteomyelitis, *July*, 87, 93
history of case, *July*, 89
infected teeth in etiology, *July*, 91
neuralgia in, cause of, *July*, 94
operations in, *July*, 91, 93
Starch injury in nutrition of infants, *Jan.*, 800
Starvation in breast feeding, *July*, 128
therapeutic, dangers of, in decomposition in infants, *Jan.*, 812
in chronic gastric ulcer, *Nov.*, 453
Status hypoplasticus in chlorosis, *March*, 982
Stenosis, aortic, digitalis in, *July*, 5
mitral, digitalis in, *July*, 5
Stomach, carcinoma of, pernicious anemia and, differentiation, *May*, 1184
causes of vomiting acting from, *July*, 58
emptying time of, in achylia gastrica, *Jan.*, 701
fluoroscopic appearance of, *July*, 211
fluoroscopy of, *July*, 199
air-bubble in, *July*, 209
barium sulphate medium for, *July*, 209
duodenal bulb in, *July*, 213
Haudek's double-meal method, *July*, 220
horizontal, *July*, 214
peristaltic waves in, *July*, 212
value, *July*, 205
in splanchnoptosis, *Jan.*, 642
increased tension on walls, as cause of gastric ulcer, *Nov.*, 442
motility in achylia gastrica, *Jan.*, 700
syphilis of, *Sept.*, 359. See also *Syphilis of stomach.*
ulcer of. See *Gastric ulcer.*
Stomach-tube, use of, in chronic gastric ulcer, *Nov.*, 461
Stomatitis, mycotic, resistance of infants to, *Jan.*, 797
Strabismus in external ophthalmoplegia, *Sept.*, 283
Stricture of rectum after hemorrhoids, *Jan.*, 671
history of case, *Jan.*, 671
physical examination, *Jan.*, 671
treatment, *Jan.*, 672
non-malignant, syphilis and, differentiation, *Jan.*, 741
Strophanthin, action of, *July*, 9
Strophanthus, action of, *July*, 8
Strychnin, action of, *July*, 9
Syndrome, Addison's, case presenting, *Nov.*, 547
Syphilis and acute disseminated myelitis, differentiation, *Jan.*, 725

Syphilis in etiology of aneurysm, *Jan.*, 677; *March*, 1023
of early general paresis, *Sept.*, 301
of general paresis, *July*, 71
of progressive muscular atrophy, *Sept.*, 297
of tabes dorsalis, *May*, 1221
infantile paralysis complicating, *Nov.*, 501, 502
of liver, *Sept.*, 237
ascites in, *Sept.*, 247
blood examination in, *Sept.*, 239
cirrhosis and, differentiation, *Sept.*, 242
classification, *Sept.*, 245
fever in, *Sept.*, 247
gummata in, *Sept.*, 243
hepar multilobulatum in, *Sept.*, 243
history of case, *Sept.*, 237
jaundice in, *Sept.*, 247
liver findings in, *Sept.*, 241
pathology, *Sept.*, 242
physical examination, *Sept.*, 238
prognosis, *Sept.*, 248
spleen in, *Sept.*, 246
symptomatology, *Sept.*, 245
treatment, *Sept.*, 248
results of, *Sept.*, 241, 242
tumor formation in, *Sept.*, 247
urinalysis in, *Sept.*, 239
of lungs, *May*, 1261
of rectum, carcinoma and, differentiation, *Jan.*, 741
of respiratory tract, *May*, 1260
diagnosis, *May*, 1263
pathology, *May*, 1260
physical examination, *May*, 1262
symptomatology, *May*, 1261
treatment, *May*, 1264
of stomach, *Sept.*, 359
diagnosis, *Sept.*, 367
gastric analysis in, *Sept.*, 367
history of case, *Sept.*, 359
hour-glass deformity in, *Sept.*, 362
pathology, *Sept.*, 365
physical examination, *Sept.*, 361
roentgen-ray examination in, *Sept.*, 362, 368
stomach tests in, *Sept.*, 362
symptoms, *Sept.*, 366
treatment, *Sept.*, 368
antisyphilitic, *Sept.*, 371
of ulcer in, *Sept.*, 373
Wassermann reaction in, *Sept.*, 367
pernicious anemia and, analogy between, *Nov.*, 519
relation of, to hemolytic anemias, *May*, 1185
Syphilitic cirrhosis of liver, clinical picture, *May*, 1279
Syphilitic cirrhosis of liver, hematemesis in, *May*, 1265, 1280. See also *Hematemesis.*
hepar lobatum in, *May*, 1279
pathology, *May*, 1278
treatment, *May*, 1281
dangers in, *May*, 1282
nutrient enemata in, *May*, 1282
surgical, *May*, 1283
infection of lungs, *May*, 1253
diagnosis, *May*, 1259, 1263
differential, *May*, 1259
gumma formation in, *May*, 1261
history of case, *May*, 1255
laboratory findings, *May*, 1257
pathology, *May*, 1260
physical examination, *May*, 1254, 1262
pulmonary tuberculosis and, differentiation, *May*, 1262
roentgen-ray examination, *May*, 1258
symptomatology, *May*, 1261
treatment, *May*, 1264
pulmonary tuberculosis, *May*, 1261
Syringe, Guyon's capillary, *March*, 1063
Syringomyelia, characteristics, *March*, 950
progressive muscular atrophy and, differentiation, *Sept.*, 295

Tabes dorsalis, *May*, 1217
after-treatment, *May*, 1235
antisyphilitic treatment, *May*, 1233
backache in, *May*, 1224
combined degeneration of spinal cord and, differentiation, *May*, 1231
diagnosis, differential, *May*, 1231
early prognosis, *May*, 1233
etiology, *May*, 1221
failure of vision in, *May*, 1224, 1226
Frankel's exercises in, *May*, 1235
gait in, *May*, 1217, 1225, 1226
histories of cases, *May*, 1219, 1224, 1226, 1227, 1229, 1230
laboratory findings in, *May*, 1225
loss of vibratory sense in, *May*, 1223
multiple neuritis and, differentiation, *May*, 1232
optic atrophy in, *May*, 1226
nerve atrophy and, differentiation, *May*, 1232
pain in, *May*, 1223
pathology, *May*, 1221
physical examination, *May*, 1220
ptosis of eyelids in, *May*, 1226
strangling in, *May*, 1220
symptoms, *May*, 1222
syphilis in etiology, *May*, 1221

Tabes dorsalis, treatment, *May*, 1233
care of teeth in, *May*, 1234
precautions in, *May*, 1234
Tachycardia, paroxysmal, *May*, 1214
cause, *May*, 1214
digitalis in, *May*, 1215
prognosis, *May*, 1215
symptoms, *May*, 1214
Tawara's node, *May*, 1201
Tea in artificial feeding, *Sept.*, 266
Teeth as foci of infection, *July*, 94
blind abscess of, relation of, to systemic diseases, *March*, 994
roentgen-ray diagnosis, *March*, 995
care of, in treatment of tabes dorsalis, *May*, 1234
contra-indications to removal of, in oral infections, *July*, 147
infections of, in streptococcic osteomyelitis, *July*, 91
radiography of, in oral infections, *July*, 138
Temperature, subnormal, in decomposition in infants, *Jan.*, 810
Test-meal, barium, for roentgen-ray examination, *Jan.*, 820
in diagnosis of achylia gastrica, *Jan.*, 702
Thoracentesis in pneumothorax, *May*, 1292
Thrombi, origin of, *July*, 100
Thrombosis in etiology of paraphasia, *July*, 102, 109
Thrombo-ulcerative endocarditis, *July*, 181
autopsy findings, *July*, 185
hemolytic jaundice in, *July*, 185
hemorrhage in, *July*, 183
history of case, *July*, 181
regional examination in, *July*, 181
Thrush, resistance of infants to, *Jan.*, 797
Toe-drop in infantile paralysis, *Nov.*, 485
Toes, gangrene of, in diabetes mellitus, *July*, 19, 25
Tonsillitis, acute nephritis following, *May*, 1307. See also *Nephritis, acute, following tonsillitis.*
in etiology of serofibrinous pericarditis, *March*, 943
Tonsils, infected, in etiology of Basedow's disease, *July*, 87
Tophus, examination of, in atypical gout, *Nov.*, 604
Trances in neurasthenia, *Jan.*, 712, 713
Transudates in ascites, *May*, 1241
Trauma, gangrene of lung from, *Nov.*, 562
Trichinosis, miliary tuberculosis and, differentiation, *Sept.*, 228
Trichomonads in protozoic enterocolitis, *May*, 1118
Trichomonas hominis intestinalis in protozoic enterocolitis, *May*, 1133
Tuberculides, *Sept.*, 319
Tuberculin test in pulmonary tuberculosis, *March*, 970
in early pulmonary tuberculosis, *Jan.*, 753
in hemopneumothorax, *May*, 1168
Tuberculosis, acute miliary, *Sept.*, 221
early pulmonary, *Jan.*, 745
accurate history-taking in, *Jan.*, 749
auscultation in, *Jan.*, 747
diagnosis, *Jan.*, 745
Koch's old tuberculin in, *Jan.*, 754
precautions in, *Jan.*, 759
with tuberculin, *Jan.*, 753
fluoroscopic examination, *Jan.*, 753
grip in etiology, *Jan.*, 749
histories of cases, *Jan.*, 746
hyperthyroidism in, *Jan.*, 757
in children, von Pirquet reaction in, *Jan.*, 754
palpation in, *Jan.*, 746
percussion in, *Jan.*, 747
physical examination, *Jan.*, 746, 751
pretuberculous stage, *Jan.*, 750
roentgen-ray signs in, *Jan.*, 754
simulated, history of case, *Jan.*, 756
physical examination, *Jan.*, 756
symptoms, *Jan.*, 750
peritoneal, retroperitoneal sarcoma and, differentiation, *March*, 1046
pulmonary, chlorosis and, differentiation, *March*, 969
diabetes mellitus with, *July*, 21
gangrene of lung and, differentiation, *Nov.*, 559
Goldscheider's orthopercussion in, *March*, 966
hemopneumothorax, *May*, 1165
in etiology of pneumothorax, *May*, 1296
inherited predisposition to, *Jan.*, 748
Krönig's apical percussion in, *March*, 966
occupation in etiology, *Jan.*, 748
skin pigmentation in, *Nov.*, 550
syphilis of lungs and, differentiation, *May*, 1262
syphilitic, *May*, 1261

Tuberculosis, pulmonary, threshold percussion in, *March*, 966
tuberculin tests for, *March*, 970
Turgor in normal infant, *July*, 113
Typhoid fever, acute miliary tuberculosis and, differentiation, *Sept.*, 225, 228
anaphylaxis in, *March*, 871

Ulcus carcinomatosum, spasm at cardia in, *Nov.*, 608
Underfeeding in etiology of decomposition in infants, *Jan.*, 809
of infants, results, *Jan.*, 801
Unverricht procedure in valvular pneumothorax, *May*, 1294
Urethritis, specific, *March*, 1057
alcoholic test-drink in, *March*, 1067
detection of gonococcus in, *March*, 1059
formation of stricture in, *March*, 1063
history taking in, *March*, 1058
involvement of Littre's glands in, *March*, 1063
of Morgagni's crypts in, *March*, 1062
method of staining gonococcus, *March*, 1061
pathology, *March*, 1061
physical examination, *March*, 1058
posterior, case, *March*, 1068
internal treatment, *March*, 1068, 1069
postgonorrheal catarrh in, *March*, 1067
simple anterior, case, *March*, 1059
treatment, *March*, 1064
dietetic, *March*, 1070
Guyon's capillary syringe for, *March*, 1063
hand injection by patient in, *March*, 1065
instruments for, *March*, 1063
local, *May*, 1196
materials in, *March*, 1063
of exacerbation in, *March*, 1067
two-glass test in, *March*, 1064
Urinalysis in chlorosis, *March*, 976
in pyelitis, *March*, 897, 900
Urine, methods of estimating acidosis applicable to, *Jan.*, 687
relations of, to artificial feeding, *Sept.*, 252
Urobilin, Schlesinger test for, *May*, 1094
Urobilinogen, Ehrlich aldehyd reaction for, *May*, 1094
Urotropin in pyelitis, *March*, 904

Vaccination in infantile paralysis, *Nov.*, 484
Vaccines, autogenous, action of, in oral infections, *March*, 992
in acne, *Sept.*, 321
Vagotonia, spasm at cardia in, *Nov.*, 613
Valvular drainage in valvular pneumothorax, *May*, 1295
pneumothorax, *May*, 1298. See also *Pneumothorax, valvular.*
Van Slyke's method of estimating reserve alkalinity in acidosis, *Jan.*, 686
Vegetables, contamination of, in etiology of protozoic enterocolitis, *May*, 1123
Venesection, digitalis with, *July*, 6
Venous pulse, newer analysis of, *May*, 1201
Ventricular extrasystole, *May*, 1205
pulse wave of Mackenzie, *May*, 1202
Vertigo in aneurysm of aorta, *March*, 1025
Virchow's theory of chlorosis, *March*, 981
Visceroptosis, *Jan.*, 636. See also *Splanchnoptosis.*
Visual aphasia, *July*, 104
Vomiting, *July*, 53
case reports, *July*, 54–56
causes, *July*, 53, 58
acting from central nervous system, *July*, 60
from stomach, *July*, 58
from various viscera, *July*, 59
study of vomitus in determining, *July*, 61
cyclic, treatment, *July*, 63
definition, *July*, 53
direct causes, *July*, 53
from acute gastritis, *July*, 58
from incomplete swallowing, *July*, 57
from kidney disease, *July*, 59
from migraine, *July*, 61
from pelvic diseases in female, *July*, 60
from prostatic hypertrophy, *July*, 60
from sea-sickness, *July*, 61
grouping of causes, *July*, 53
in carcinoma of esophagus, *May*, 1151
in cardiospasm, *Nov.*, 620
in gastric ulcer, *Nov.*, 420
in pregnancy, *July*, 60
in stenosis of pylorus and cholelithiasis, *Jan.*, 786, 790
indirect causes, *July*, 53, 54
management of, *July*, 64
mechanism of, *July*, 57
nervous, treatment, *July*, 62
regurgitation of food and, differentiation, *July*, 56
rôle of nausea in, *July*, 61

Vomiting, rumination and, differentiation, *July*, 57
selective, treatment, *July*, 62
so-called, in infants, *July*, 57
symptomatic nature of, *July*, 54, 56
time of occurrence, *July*, 61
treatment, *July*, 62–64
Vomitus, study of, in determining cause of vomiting, *July*, 61
von Jaksch's anemia, *May*, 1181
pernicious anemia and, differentiation, *Sept.*, 405
splenectomy in, *Sept.*, 416
von Noorden's theory of chlorosis, *March*, 983
von Pirquet reaction in early tuberculosis in children, *Jan.*, 754

Wassermann reaction in syphilis of stomach, *Sept.*, 367
Water-supply in etiology of protozoic enterocolitis, *May*, 1122
Werlhof's disease, *Jan.*, 849. See also *Purpura hæmorrhagica.*
Wernicke's conduction paraphasia, *July*, 103
speech tract, *July*, 108
Wickmann's classification of infantile paralysis, *Nov.*, 505
Widal and Chauffard's test for fragility of red blood-cells, *May*, 1095
Wilson's disease, paralysis agitans and, differentiation, *March*, 951, 958
Word-blindness, *July*, 104
Woulff bottle exercise in hemopneumothorax, *May*, 1173
Wrist-drop in lead-poisoning, *Sept.*, 378

x-ray. See *Roentgen ray.*

The Medical Clinics of Chicago

VOLUME 2

1916-1917

PHILADELPHIA AND LONDON

W. B. SAUNDERS COMPANY

COPYRIGHT, 1916 AND 1917, BY W. B. SAUNDERS COMPANY. ALL RIGHTS RESERVED

PUBLISHED BI-MONTHLY (SIX NUMBERS A YEAR), BY W. B. SAUNDERS COMPANY, WEST WASHINGTON SQUARE, PHILADELPHIA

CONTRIBUTORS TO VOLUME 2

ISAAC A. ABT, M. D.

Professor of Diseases of Children, Northwestern University Medical School; Attending Physician, Michael Reese Hospital (Sarah Morris Memorial Hospital for Children).

ARTHUR F. BEIFELD, M. D.

Instructor in Medicine, Northwestern University Medical School; Attending Physician to Cook County Hospital, etc.

TRUMAN W. BROPHY, M. D., D. D. S.

President and Professor of Oral Surgery, Chicago College of Dental Surgery; Oral Surgeon, St. Joseph's and Michael Reese Hospitals, etc.; Consulting Oral Surgeon, Presbyterian Hospital.

JAMES T. CASE, M. D.

Professor of Roentgenology, Northwestern University Medical School; Roentgenologist to St. Luke's Hospital, Chicago, and to the Battle Creek Sanitarium, Battle Creek, Michigan; Consulting Roentgenologist, Cook County Hospital.

B. C. CORBUS, M. D.

Instructor in Genito-Urinary Surgery, Rush Medical College.

ARTHUR R. EDWARDS, M. D.

Professor of Principles and Practice of Medicine, Northwestern University Medical School; Attending Physician, Michael Reese Hospital, etc.

CHARLES A. ELLIOTT, M. D.

Assistant Professor of Medicine, Northwestern University Medical School; Attending Physician, Wesley Memorial Hospital.

JOSEPH C. FRIEDMAN, M. D.

Assistant Professor of Medicine, Northwestern University Medical School; Attending Physician, Michael Reese Hospital.

WALTER W. HAMBURGER, M. D.

Assistant Professor of Medicine, Rush Medical College; Attending Physician, Cook County Hospital, etc.

RALPH C. HAMILL, M. D.

Assistant Professor of Nervous and Mental Diseases, Northwestern University Medical School; Attending Neurologist, Cook County Hospital, etc.

HERMAN L. KRETSCHMER, M. D.

Urologist, Presbyterian Hospital; Assistant in Genito-Urinary Surgery, Rush Medical College.

CHARLES LOUIS MIX, M. D.

Professor of Physical Diagnosis, Northwestern University Medical School; Attending Physician, Mercy Hospital.

M. MILTON PORTIS, M. D.

Assistant Professor of Medicine, Rush Medical College; Attending Physician, Cook County Hospital, etc.

WILLIAM ALLEN PUSEY, M. D.

Professor of Dermatology, Medical Department, University of Illinois; Dermatologist to St. Luke's and Augustana Hospitals, etc.

H. H. SCHUHMANN, M. D., D. D. S.

Oral Surgeon to Michael Reese Hospital.

FRANK SMITHIES, M. D.

Gastro-enterologist to Augustana Hospital, Chicago; formerly Instructor in Internal Medicine and Demonstrator of Clinical Medicine in the University of Michigan, Ann Arbor.

DAVID C. STRAUS, M. D.

Assistant in Surgery, Rush Medical College; Associate Attending Surgeon, Michael Reese Hospital, Chicago.

SIDNEY STRAUSS, M. D.

Assistant Professor of Medicine, Rush Medical School; Assistant Attending Physician to Presbyterian Hospital and Michael Reese Hospital.

SOLOMON STROUSE, M. D.

Instructor in Clinical Medicine, University of Illinois Medical School; Associate Attending Physician, Michael Reese Hospital, etc.

FREDERICK TICE, M. D.

Professor of Diseases of Chest and Clinical Medicine, Medical Department, University of Illinois; Attending Physician, Cook County Hospital, etc.

CHARLES SPENCER WILLIAMSON, M. D.

Professor of Medicine and Clinical Medicine, Medical Department, University of Illinois; Attending Physician, Cook County Hospital, etc.

FRANK WRIGHT, PH. C., M. D.

Assistant Professor of Physiological Chemistry, Northwestern University Medical School; Associate Attending Physician, Michael Reese Hospital.

JOSEPH ZEISLER, M. D.

Professor of Skin and Venereal Diseases, Northwestern University Medical School; Attending Dermatologist, Wesley and Mercy Hospitals; Consulting Dermatologist, Michael Reese Hospital.

RC
39
M38
v.2
no.6

The Medical clinics of Chicago

Biological
& Medical
Serials

PLEASE DO NOT REMOVE
CARDS OR SLIPS FROM THIS POCKET

UNIVERSITY OF TORONTO LIBRARY